A PELICAN BOOK

DONALD HUNTER

Health in Industry

Diseases and accidents
to which workers in industry
are liable, and how they
may be treated or prevented

A VOLUME OF THE
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Occupational diseases have a long history and terms such as housemaids' knee, grocers' itch, and writers' cramp have been in use for many years. In the eighteenth century the Industrial Revolution brought great prosperity to England but it also brought industrial disease to factory, workshop, and mine. It was natural that it should have been English doctors who first took responsibility for the health of the worker in industry. But in spite of the Factory Acts progress was slow, and new problems have continued to arise.

The author's main aim is to emphasize that in this field prevention is better than cure, and it is in this interest that architects and ventilating engineers cooperate with doctors and chemists in a vast programme of preventive medicine. The work of the different agencies concerned with the problems of health in industry is examined, and chapters deal with such subjects as accidents, poisoning, dust diseases of the lungs, and many other occupational hazards. There is a glossary which will help the general reader to understand the technical terms, and a useful bibliography; the book is illustrated by eight pages of plates.

PELICAN BOOKS

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TO LIZZIE

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EDITORIAL FOREWORD

HEALTH is hard to define. To say that it is absence of disease just 'won't do'. For any satisfactory definition of disease is equally, if not more, difficult. Rather, let us say that it is that general sense of physical well-being combined with successful adaptation of the mind to the recurring problems of human existence to which most people feel instinctively that for some reason they are entitled. Health, as thus defined, comes naturally to many, but it can as we all know be only too easily either temporarily or permanently lost.

On what does health depend? On many conflicting factors: in part on the genetic constitution of the man, i.e. on the plan for his body-mind which was laid at his conception and medical science cannot alter that; in part on the circumstances, physical and psychological, under which he grew up, very much indeed on the influence of home and school and university. It depends, also, on personal effort and deliberate way of life; in fact on the way in which the mind developed on, and conditioned by the body makes use of the body on which it is based. (So many seem deficient in the art of living!) It also depends on a man's work and that may be determined by his environment on circumstances or by tradition; by advice given to him, either good or bad; or by deliberate self-choice, by no means always right in relation to his genetic constitution and his upbringing.

This latest Pelican medical book is therefore particularly important for the simple reason that it discusses health in relation to the occupation of the vast majority of people in this country, indeed the world over, namely work in industry of some kind. Further, its title is significant, reflecting the hopeful modern trend. Had Dr Donald Hunter been writing a generation ago he might (although, knowing the man, I very much doubt that he would) have called his book *Disease in Industry*, reflecting the defeatist attitude of the doctor to disease in general, and to disease in industry in particular, prevailing at that time. But not so today. Dr Donald Hunter writes hopefully of *Health in Industry*. True that in a succession of fascinating chapters he does not hesitate to paint the picture of industrial risks and describe without fear or favour the many lurid patches in medical history which accompanied the industrial revolution, partly due to the grim philosophy of the time, partly to inevitable uneconomic competition, partly to over-rapid mechanical invention, and partly to the medical ignorance then prevailing. But the whole spirit of his book is that disease in industry

has been, is being, and will be still further prevented. Health in industry is in point of fact being achieved. This country set the pace in industrial development and paid, it is true, a heavy price for her rapidly increasing wealth in the expensive currency of individual sickness and disease. But it is also true, as the author is at pains to point out, that England by successive Acts of Parliament also set the pace in the reform and regulation of the hours and conditions under which men and women are allowed to work in industry today in every civilized country of the world.

Risks must be taken in most walks of life however. Half the art of living, indeed, is to be prepared to live life dangerously, and the distinguished author of this book would be the first to subscribe to this philosophy. Nevertheless, risks should and can be reduced to a minimum and Dr Donald Hunter tells us how much has been done in this direction already and points the way to how they can be reduced still further in the future. But, as he tells us, particularly in the early chapters of this book, health in industry depends on something much more than that. It also depends on happiness in industry, and happiness in turn depends on getting the right man into the right job, giving him self-respect, security, and responsibility, adapting his work, when necessary, to acquired disability or advancing age and fostering that spirit of fair play and mutual cooperation between employers and employed which is so necessary to efficiency in any branch of industry. So this book should be read, not only by medical students and doctors, but in its less technical parts also by social workers and almoners, employers of labour and trade union officials, Members of Parliament and politicians. For its approach is such that it will do much to foster that peace and harmony in industry on which the wealth of the nation and, indeed, that of the whole world depends.

Finally, may I be allowed to conclude on a personal note? I take pride in the fact that I succeeded in persuading my friend and colleague, who with boundless human sympathy and understanding has succeeded in combining life in consulting practice with the intensive study of disease in industry, to write this book. It has been a privilege to have had this opportunity to introduce it to the reader.

A. E. CLARK-KENNEDY.

PREFACE

THE Industrial Revolution in the eighteenth century gave rise to discomforts and diseases which did not affect the privileged classes of society but only those who laboured in workshop, factory, or mine. For a long time the work of these men, which was indispensable to the prosperity of the community, brought them nothing but physical, intellectual, and moral poverty. And then men like Albert Thomas awakened the conscience of the modern world to this monstrous social paradox. Too much misery lies behind many humble daily tasks, they lead to much unnecessary physical, and hence also moral suffering. It was Pierre Hamp who pointed out that we live on the sufferings of others. Everyone makes life a torment for some of his fellow-men. How many people earn their living pleasantly? Many do so in unpleasant and nearly intolerable conditions. To love one's occupation is to be happy, but where are the occupations which one can love?

From the time of Hippocrates onwards the existence of specific occupational diseases has come to be recognized in our common speech in such expressions as brassfounders' ague, chimney-sweeps' cancer, divers' paralysis, glass-blowers' cataract, grocers' itch, hatters' shakes, housemaids' knee, knife-grinders' phthisis, miners' nystagmus, painters' colic, tailors' callosities, wool-sorters' disease, and writers' cramp. A certain number of trades are recognized by the law to be dangerous, but there are far more without toxic hazards which are uncongenial and stifle the creative urge. When sickness rates, accident rates, and absenteeism are exceptionally high in a particular form of industry then it becomes highly probable that much of this is dramatized discontent and that that particular form of work is, by its nature, or because of the conditions which attend it, itself disease-provoking. Since one of the main social functions of medicine is to prevent and to control the action of disease-provoking agencies it follows that this is a matter which attracts the attention of doctors.

Undergraduates in the faculties of medicine, science, arts, or law who plan a career connected in any way with industry, may find in this book information and ideas relevant to their future work. But I hope that it will be found useful by others such as general practitioners, medical consultants, personnel officers, works chemists, works managers, trade union officials, welfare workers, industrial nurses, and indeed all those whose task it is to see that a particular factory or

workshop is a fit place in which men and women can work in health and happiness. I have found impossible the task of writing the book in terms intelligible to those with no training in chemistry, physics, biology, or medicine. But fortunately more than half the book is free from technical terms, and it is hoped that the general public may derive pleasure and instruction from it by passing over what is highly technical and by using the glossary at the end.

Of the world's workers university graduates in science and medicine are to be numbered among the fortunate, for they do jobs of their own choosing. Moreover their work gives them abiding satisfaction and they are permitted to think that what they do contributes to the common weal. It is with understanding and sympathy that a doctor who is so favoured does what he can to help those whose work is unpleasant or monotonous. This book is written in that spirit.

Whitechapel, E.1
June 1958

DONALD HUNTER

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Plate 16 is reproduced by kind permission of the United Kingdom Atomic Energy Authority. For all the other plates, which are taken from his book *The Diseases of Occupations* (1957), the author is indebted to The English Universities Press, Ltd.

CHAPTER ONE

INTRODUCTORY

Distribution of Manpower – The Working Environment – Training the Medical Student – Training the Industrial Medical Officer

SOCIALLY and historically the conditions and life of the industrial worker lie near the foundation of the well-being of an industrial people. In England that vital issue was raised at the beginning of the Industrial Revolution in the middle of the eighteenth century, and it has remained with us, increasing in magnitude and complexity as the population and the various industries have expanded. Its development is one of the most impressive in our social annals. Its lessons of triumph and failure have been carried to the ends of the earth. For England was the first of the civilized nations to embark, all unwittingly, upon a rapid and unforeseen evolution of industrial enterprise. In some ways she has been surpassed by her foreign competitors, but she remains the originator of mechanical invention by a whole people. For it was England which first learned the great principles of restricting the hours of labour, of safeguarding the health of the factory worker, of exploring the effect of occupation upon health, and of the prevention of its ills and accidents. It was England also which contrived methods of State control in the form of the Factory Acts.

DISTRIBUTION OF MANPOWER

In Great Britain, twenty-four million men and women earn their daily bread by manual labour or otherwise. There are also about ten million housewives who work hard in the home but are not paid. The twenty-four million workers are scattered through many occupations and include those serving with the armed forces, those in industry, those on the land, those in professions, and the registered unemployed. Table I shows the distribution of the working population in April 1958.

TABLE I

Distribution of Manpower in Great Britain, April 1958
(*Ministry of Labour Gazette*, June 1958)

	Thousands
Total Working Population	24,068
Armed Forces (including leave)	622
Mining and Quarrying	862
Gas, Electricity, and Water	380
Transport and Communications	1708
Agriculture and Fishing	967
Manufactures	9203
Building and Contracting	1472
Distributive Trades	2965
Professional, Financial, and Miscellaneous Services	4204
National Government Service	530
Local Government Service	755
Total in Civil Employment	23,046
Unemployed	442

The basic industries and services are mining and quarrying; gas, electricity, and water; transport and communications; and agriculture and fishing. On these, the country depends for its food, the rest of its industrial and manufacturing activities, and much of its daily life. Without coal, there would be no steam, gas, or electricity; without water, sanitation and drainage would break down; without fishing and agriculture there would be inadequate food; without quarrying, little building; and without transport and communications our complicated economic life would come to a stop. These industries serve the manufacturing industries. They move the machinery, light the workshops, bring raw materials to the factories, and distribute the finished goods.

Although the adult male in the prime of life is generally the most efficient worker, there are certain jobs for which he is not particularly suited and, further, there are insufficient men in their prime to do all the industrial work needed. For these and other reasons, young persons, women, and old people are employed in industry in large numbers. Women form less than one-third of all industrial workers. In the basic industries of mining and quarrying, supply services, transport and communications, where much of the work is heavy and shift work is common, very few women

are found. In building there are even fewer, and in the heavy metal and chemical trades they comprise less than one-fifth of the labour force. Half the workers in the distributive trades are women, mostly shop assistants. The textile and clothing industries employ more women than men.

The Working Environment

The health and happiness of the worker in industry depend on circumstances which are as complex as they are numerous. Experts in widely differing fields are constantly at work to secure the best results. *Architects* must be employed so that industrial firms may set out to design factories which are a joy to work in rather than being just tolerable. *Ventilating engineers* are needed to maintain comfort in working and in the case of the dangerous trades to keep the working atmosphere free from poisonous vapours, fumes, and dusts. *Chemists* will help in this work, and wherever there is a hazard from radioactive substances, *physicists* will be consulted. *Illumination engineers* will be required for innumerable jobs, and of course their contribution to safety is considerable. *Mechanical engineers* will devise machines which are fool-proof against amputating people's fingers, and *electrical engineers* will arrange switching systems making machines fool-proof against electrocution accidents. *Safety officers* will provide protective clothing from plastic helmets to boots with steel toe-caps. The Works Safety Committees will hold discussions on accident prevention and arrange propaganda devices in such forms as posters and competitions. And then *factory inspectors* will come to see that all the regulations are obeyed. They may seek the assistance of *medical inspectors*, *electrical inspectors*, or *engineering and chemical inspectors*. *Personnel managers* and *welfare workers* will be responsible for incentive schemes. *Statisticians* will contribute to the study of health in industry by arranging punched-card systems to record sickness absence in groups of employees. In this the London Transport Executive (1956) has led the way by setting up a Central Record of Staff Statistics at its headquarters.

The medical department will be in charge of the *works medical*

officer assisted by *nurses* specially trained for the job and perhaps *physiotherapists. Dental surgeons* may attend regularly for the benefit of the workers. The works doctor will supervise the training of squads of *first-aid men* in rescue work and they will keep all the special breathing apparatus and other rescue gear constantly in working order. *Dietitians* may be employed to arrange the menus in the works canteens and they may consult *canteen advisers.* The quality of the medical work which the works doctor has to offer will always be of paramount importance. The actual impetus for improved conditions at work has largely come from doctors and will continue to do so. Clearly the doctor in training must understand that the factors concerned in keeping the worker healthy are more than medical, they are social, economic, environmental, political, psychological, and personal. How is this brought about in our medical schools?

Training the Medical Student

Let us begin by considering some points in the training of students in the medical schools of Great Britain. What they have to learn is difficult and complex and necessitates long concentration on bedside methods in which they are taught how to question and examine patients. In doing this they learn to take an interest in patients as men and women who lead a particular kind of life in a certain kind of way, as persons with family ties and obligations, with anxieties, hopes, and fears. A good doctor takes into account social and economic factors, conditions of work and leisure, standards of housing, clothing, diet, and personal habits. He sees the injured worker as a bread-winner, the woman in childbirth as a wife and mother, the handicapped child as an educational problem and a source of special anxiety for the parents. Hence, from the beginning of his training, the medical student should be led to embrace the notion of a diagnosis which relates both the physical condition and the patient himself to the environment in which he lives, works, and plays. The environment of the patient may be thought of as psychological, occupational, and socio-economic.

The social aspects of the doctor's work are today linked very

closely with the positive promotion of health and the prevention of disease. The effective treatment of occupational diseases is essentially preventive. Often in undergraduate medical education the role of social and preventive medicine is too little emphasized. In some medical schools the undergraduate receives inadequate instruction in matters upon which health largely depends: nutrition, housing, factory hygiene, personal hygiene, socio-economic status, social security, and organized public health and medical-care services.

A medical graduate with nothing but a bedside training might very well practise medicine without ever having asked himself: 'How much of this disease is there? In what circumstances is the incidence high or low? How could it be prevented?' He might never have formulated the questions: 'What social aids are needed to reinforce medical care? How completely and in what manner are they provided? What is my part as a practising doctor in the medical welfare services maintained by the State and other agencies? What are the wider duties in society of the profession to which I belong?' There can be little doubt about the vocational and educational inadequacy of medical training which does not raise, examine, and point the way to answering these and other questions of like nature.

Should the teachers of medicine arrange to take medical students on visits to factories, docks, and mines? Undoubtedly they should, but too often they do not. The pressure of other duties is no doubt their excuse. A visit to sewers is a single example of what may be done. Teachers in any medical school in Great Britain can arrange with the main drainage engineers of the local authority to walk with their students through a section of the sewers of the town in which they work. And when the student becomes a qualified doctor he should take the opportunity to enrich his general knowledge and experience by studying the conditions of work in those industries which fall within the area of his practice. By visiting the places where his patients work not only will he understand their problems better, but also he will be able to help them by making contact with the welfare and medical departments within the factories concerned. No doctor can be expected to be familiar with the details of all occupations and

every working environment, but at least he should learn how to obtain the requisite information from libraries.

Training the Industrial Medical Officer

In Great Britain there are fewer than 3000 medical men and women engaged in one way or another in industrial medicine and only about 250 who give their whole time to it. There are approximately 250,000 factories in the country and of these 230,000 employ fewer than 250 persons each, many as few as 25 persons. Of those factories with 250 employees only 50 per cent employ a doctor, and where there are fewer than 100 workers only 4 per cent have the services of a doctor.

To qualify as competent to carry out the duties of full-time industrial medical officer a doctor will follow a prolonged course of study for a special diploma, usually of a university. The details need not be given here, but let it suffice to say that he must be prepared to make himself technically minded to a degree that used to be thought quite foreign to the sphere of practical medicine.

Lighting, heating, ventilation, humidity, dust control, prevention of emission of toxic gas and fume, optimum methods of working, weight lifting, shift systems, ways of avoiding boredom in repetitive work, adjustment of human relations, detection and removal of sources of friction and fear, investigation of processes known or suspected to lead to disease, development of physical and chemical methods to determine the absorption by the worker of dangerous compounds, maintenance of high standards of hygiene and sanitation, control of nutritional standards in canteens, development of the best technique for the treatment of injuries and poisoning – all come within the scope of environmental study, in the factory and in the laboratory. By such detailed study of industrial environment he appreciates the background against which to place new entrants, for rehabilitating the injured and partly disabled, for choosing alternative work for a man who has been sick, and for elucidating possible causes of acute and chronic illness among the workers.

The full-time industrial doctor of the future must be so well trained that he will be invited to co-operate with managers,

workers, engineers, chemists, and architects. He should aim at discovering all possible faults in the working environment with a view to finding proper remedies for them. One of his most interesting duties must be to bring into the effective service of industry the discoveries of the research worker. Only then will he be in a position to make the industrialist understand the risks to which his men are exposed, and that the guiding principle of low costs and large profits retards progress. He should have an aptitude for administration, though in the factory he is best employed in an advisory, not an executive, capacity. He should be responsible to the managing director, and not to any other official of the company, such as the labour manager. He should take charge of all first-aid, medical, and nursing services. He should have had considerable experience in clinical work and should have held resident hospital appointments; he should know something of social problems and should be able to undertake original research in medicine. The factory medical officer should never allow his special technical knowledge to assume greater importance than his knowledge of doctoring in general. He should strive to be a good doctor in the broadest sense of the term, to preserve contacts with academic medicine, to cultivate interests outside his profession, and to follow his various activities with a constant regard for social values.

In modern industrial medicine the emphasis is upon people, the conditions in which they live and work, their hopes and fears, their abilities, their attitudes towards their job, their fellow workers, and their employers. Of course, the industrial doctor must have a practical clinical background which will enable him to assess physical factors in health and disease, and detect disease attributable to occupation. Far more difficult is the assessment of men and women as emotional beings, moved by trivial things, overturned by worries and anxieties, torn by conscience, stunned by the inevitable trials of family life, and easily captured by a sense of frustration and persecution. It is the interaction of this complex, an individual and his industrial environment, animate and inanimate, that the industrial doctor tries to analyse, and from the analysis to establish an equilibrium which permits a stable relationship.

As social-mindedness develops, a better industrial medical service will be demanded. At present the supply of competent doctors is inadequate to fill the number of posts which already exist in factories. Meanwhile the majority of our medical schools and teaching hospitals are not alive to this need.

CHAPTER TWO

HISTORY OF INDUSTRIAL MEDICINE

Slavery and Forced Labour in the Mines of Antiquity – Greek Prejudice Against Manual Labour – Early Occupational Medicine – The Father of Occupational Medicine – The Industrial Revolution – The Birth of Industrial Medicine in England – The Factory Inspectorate – Thomas Morison Legge, 1863–1932

THIS chapter depicts something of the historical, social, and economic background of the occupations men follow. The historical approach is necessary if we are to appreciate what has been achieved as well as what yet requires to be done.

SLAVERY AND FORCED LABOUR IN THE MINES OF ANTIQUITY

We know that the ancient Egyptians devoted great enterprise to the winning of gold, both by conquest and by mining. The Greek historian Diodorus Siculus has left us a detailed account of the horrors in the Nubian gold mines under the Ptolemies. The miner of antiquity was almost always a slave. The slaves were recruited from condemned criminals, prisoners-of-war and, when the supply of workers ran short, forced labour was employed. Innocent individuals, sometimes entire families or tribes, were exiled to the mines. The miners worked in chains, practically naked and guarded by foreign mercenaries with whom they were unable to talk. The lash of the overseer drove them to their arduous labour. The workers were divided into three groups; the strongest adult men did the actual work of breaking the rock, and handled pick and pitching-tool; children carried the broken ore out of the mine, and the women and old men were employed to grind it by hand. When the rock was too hard to be attacked with quarrying tools, the method of fire-setting was employed. This procedure, which is also mentioned by Pliny and other writers of antiquity, consisted in placing a fire against the ore face and then pouring

water on the heated rock. On cooling rapidly, the rock cracked and was easily broken loose with the usual tools. The implements employed were of bronze and stone.

In Greek mythology Hephaestus was the god of fire and became the divine smith and patron of craftsmen. His cult reached Athens about 600 B.C. In epic poetry he is a pathetic figure, lame and unkempt in appearance. In Homer his skill in metallurgy is often mentioned, his forge being on Olympus, where he is served by images of golden handmaids whom he has animated. Vulcan the Roman god of iron, brass, and devouring flame also was ugly, misshapen, and lame. He was the most industrious of all the gods, but whether he was the deity of smiths is doubtful. Perhaps he was too busy making jewels for goddesses, thunderbolts for Jove, and weapons for Achilles to be of much help to humbler craftsmen. The legendary genealogy of Tubal-cain is recorded in the Old Testament. Lamech, a descendant of Cain, had three sons, Jabal, Jubal, and Tubal, to whom are attributed the development of pastoral civilization, music, and metalwork respectively. Myths similar to those of ancient Greece and Rome are found in relation to the Teutonic Wieland who also was lame, to the Scandinavian Volundr, lord of the elves, and to the Finnish Smith-god Ilmarinen. English legend makes Wayland the Smith the mythical hero of the skilled metal worker; local tradition has placed his forge in a cave close to the White Horse in Berkshire.

Greek Prejudice Against Manual Labour

Studies in social history by Professor Benjamin Farrington (1941) have revealed an attitude of prejudice which existed in ancient Greece towards those citizens who plied a mechanical trade. This attitude to manual labour led to a decline in social status of the manual labourer which accompanied the growth of civilization.

He pointed out how Xenophon in his treatise called *Oeconomicus* represented Socrates as delivering the following judgement on manual work and the manual worker.

> What are called the mechanical arts, carry a social stigma and are rightly dishonoured in our cities. For these arts damage the bodies of those who work at them or who have charge of them, by compelling

the workers to a sedentary life and to an indoor life, by compelling them, indeed, in some cases to spend the whole day by the fire. This physical degeneration results also in deterioration of the soul. Furthermore, the workers at these trades simply have not got the time to perform the offices of friendship or of citizenship. Consequently, they are looked upon as bad friends and bad patriots. And in some cities, especially the warlike ones, it is not legal for a citizen to ply a mechanical trade.

Obviously a social division so deep as this, a cleavage which when complete made it impossible for the same man to be both worker and citizen, could not be without effect on the science and practice of medicine, which touch the life of every man. But the nature of this effect remained inadequately explored until Professor Farrington undertook the task. He discussed fully the limitations of ancient medical science and practice in respect of the type of person and the type of disease it habitually dealt with and habitually neglected. Roughly speaking, the working man was neglected in ancient medical practice and the occupational diseases ignored in medical science.

Undoubtedly Hippocratic medicine was limited in its application to a section of the people. A treatise like *Airs, Waters, Places* was written for citizen doctors with citizen patients in view, and those, too, of the leisured class. The Hippocratic medicine, we are informed by all competent inquirers, rested on the concept of a balance between the living organism and its environment. It regarded sickness as an effort to restore a disturbed equilibrium, and the duty of the physician was to co-operate with nature in her efforts to secure a readjustment. Therefore the Hippocratic doctor, who was frequently, perhaps even normally, itinerant, was taught to study, as he came to each new locality, the major features of the environment of his future patients.

This is the subject of *Airs, Waters, Places*. As the title indicates, it was the natural features of the place he was taught specially to observe – the climate, the situation, the quality of the water. He was also given hints as to the kind of constitution he might expect to find in the inhabitants of a town living under conditions of oriental despotism as contrasted with those enjoying the privileges of Greek liberty. That is to say, even the political environment of

a patient was to be taken into consideration by the Hippocratic doctor. Historians have been very properly impressed with the comprehensive outlook of the ancient medical manual. But, if we look at it carefully, we can see that it is deficient, professing to be a treatise on environment and yet omitting what may be described as the most important element in the environment from the point of view of health and disease, the regular occupation of the man.

If we turn to the four books of the Hippocratic treatise called *Regimen*, we find that the author of this much admired and very important treatise developed a theory that health depends upon a balance of food and exercise. But the foods which he discusses hardly suggest the diet of a potter or a peasant, and the exercise which he recommends has nothing to do with work. Thus it would be a mistake to suppose that beef, hare, fox, and hedgehog all formed a normal part of the diet of the working man, slave or free, any more than doves, partridges, pigeons, cocks, turtles, geese, ducks, and other marsh or river fowl.

And it would equally be a mistake to suppose that the following counsels as to exercise were addressed to the working man:

> Exercises should be many and of all kinds; running on the double track increased gradually; wrestling after being oiled, begun with light exercises and gradually made long; sharp walks after exercises, short walks in the sun after dinner; many walks in the early morning, quiet to begin with, increasing till they are violent and then gently finishing.

The following advice also seems not to be addressed to the worker:

> These patients ought to take their baths warm, to sleep on a soft bed, to get drunk once or twice but not to excess, to have sexual intercourse after a moderate indulgence in wine, and to slack off their exercises, except walking.

But Hippocrates was not alone in his neglect of the working man; the possibility that occupational factors could be of importance in explaining a given illness was ignored all through the Dark Ages. More than 2000 years were to elapse before the Revival of Learning brought this idea to the notice of men. It occurred as a revolutionary innovation made by Ramazzini, the Father of Occupational Medicine. In his *De Morbis Artificum*

Diatriba (1713) he made a striking addition to the art of diagnosis, giving to doctors the simple practical advice that to the questions recommended by Hippocrates they should add one more, to ask the patient to describe his occupation.

Early Occupational Medicine

Throughout the medieval period there were no contributions to the subject of occupational diseases, and it is not until the sixteenth century that we find definite information relating to diseases of miners and of workmen in dangerous trades. It was in the middle of the sixteenth century that two remarkable men, Agricola and Paracelsus, wrote on the subject of miners' diseases, and at the close of the seventeenth century came the classical work of Ramazzini.

In 965 silver was discovered near Goslar and mining began in the Harz mountains. Between 1100 and 1300 important discoveries of precious metals were made in the Erzgebirge. During the thirteenth century silver began to be mined in Bohemia and gold in Silesia, and for 300 years mining became an important industry in Central Europe. This meant that the Germans supplied Europe with the precious metals needed for currency, and as a result they became the leaders of the commercial world. The mines of Schneeberg were opened in 1410, and in 1516 rich veins of silver were discovered at Joachimsthal on the southern slopes of the Erzgebirge. In Bohemia in 1519 a silver coin worth 3 marks, about 2s. 11d., was minted at Joachimsthal. It became known as the *Joachimsthaler* and later as the *Thaler*. The word was corrupted in English to *daller* and about 1553 to *dollar*.

The classic description of metalliferous mining in Central Europe in the sixteenth century is to be found in *De Re Metallica* by Georg Bauer, who was more commonly known as Georgius Agricola. He was born at Glauchau in Saxony at the very beginning of the Revival of Learning. The printing press had been first used forty years before, Luther was a babe of one year, Erasmus was still a student, Columbus had just discovered America, and three years were to elapse before Vasco da Gama rounded the Cape of Good Hope. At the age of twenty Agricola went to

Leipzig to study Latin and Greek for three years, and he then taught these subjects in a municipal school in Germany. Between the ages of thirty and thirty-three he studied philosophy, medicine, and the natural sciences in Italy, attending the universities of Bologna, Venice, and perhaps Padua. In 1526 he was appointed official physician to the mining town of Joachimsthal.

De Re Metallica was published in 1556, a year after the death of Agricola. It is a scholarly work consisting of twelve books which deal with every aspect of mining and with the associated smelting and refining of gold and silver. The fifth book deals with the actual mining underground. The art of surveying in the mine, the different kinds of ores to be found, the types of shafts and how to sink them are all fully described. The sixth book describes the various tools and implements fully. It gives a complete account of the machinery employed for ventilation, pumping, and winding. Drainage was effected by means of a continuous chain of buckets operated by a foot tread-wheel. The book ends with an account of the diseases and accidents prevalent among the miners and the means available to guard against them.

The ill effects of poor ventilation were known from practical experience. Agricola says:

> Miners pay the greatest attention to these matters just as much as to digging, or they should do so. Air indeed becomes stagnant both in tunnels and in shafts. I will now speak of ventilating machines. If a shaft is very deep and no tunnel reaches to it, or no drift from another shaft connects with it, or when a tunnel is of great length and no shaft reaches to it, then the air does not replenish itself. In such a case it weighs heavily on the miners, causing them to breathe with difficulty, and sometimes they are even suffocated and burning lamps are also extinguished. There is therefore a necessity for machines which enable the miners to breathe easily and carry on their work.

Several types of ventilating machines were employed to force air into the mine workings. Agricola speaks of powerful blowing machines and he mentions the use of bellows. He also describes another ventilating machine consisting of a cylindrical barrel, within which four wings rotated.

Accidents were by no means rare. Miners slipped from the ladders into the shafts and broke their limbs or their necks, or

they fell into the sump at the bottom of the shafts where they were drowned. Falls of ground imperilled their lives, and major tragedies sometimes occurred, as on the occasion when Remmelsburg near Goslar subsided, killing 400 men. The actual methods of mining ore had changed very little from the days of ancient Egypt, except that the hammers, picks, blocks, and wedges were of iron instead of stone or bronze. Where these tools were of no avail, the method of fire-setting was used. Agricola was well aware that fire-setting had its perils.

> While the heated veins and rock are giving forth a fetid vapour and the shafts or tunnels are emitting fumes, the miners and other workmen do not go down in the mines lest the stench affect their health or actually kill them. The Bergmeister in order to prevent workmen from being suffocated gives no one permission to break veins or rock by fire in shafts or tunnels where it is possible for the poisonous vapours and smoke to permeate the veins and pass through into neighbouring mines.

In the last part of his sixth book Agricola discusses those ailments which attack the joints, the lungs, and the eyes of miners. The accounts he gives are rambling and lack precision; it is clear that the idea of diseases specifically caused by particular occupations had not, in his day, entered men's minds. His description of the harmful effects of the dust inhaled is of a suppurating disease of the lungs with a visible and progressive emaciation. It is probable that silicosis, tuberculosis, and carcinoma of the lung were involved in the conditions he describes.

> On the other hand some mines are so dry that they are entirely devoid of water, and this dryness causes the workmen even greater harm, for the dust, which is stirred and beaten up by digging, penetrates into the windpipe and lungs, and produces difficulty in breathing and the disease which the Greeks called asthma. If the dust has corrosive qualities, it eats away the lungs, and implants consumption in the body. In the mines of the Carpathian Mountains women are found who have married seven husbands, all of whom this terrible consumption has carried off to a premature death.

To protect the miners against dust Agricola advises purification of the air in the mine by ventilating machines and the use of loose veils over the faces of the miners.

In Agricola's day it was not considered unnatural for the miners to come across demons in the mine workings. These were usually considered to be jolly and of kindly intent rather than cruel or evil. They were either little boys or dwarfed men, and they did little harm beyond chattering and extinguishing the miners' lamps. Agricola wrote of them in his *De Animantibus Subterraneis* and told how demons of ferocious aspect were expelled and put to flight by prayer and fasting. In his great work *Deutsche Mythologie* (1835), Jacob Grimm (1785–1863), the philologist, perpetuated the folk-lore which surrounded these demons of the mines. In the enchanting *Kinder- und Hausmärchen* (1812), which he wrote with his brother Wilhelm, he relates the story of the persecuted child princess befriended by dwarfs who lived in the woods and worked in the mines nearby. In our time this story has been delightfully rendered into animated cartoons by Walt Disney in his *Snow White and the Seven Dwarfs*.

In 1567, eleven years after the publication of Agricola's treatise, there appeared the first monograph devoted to the occupational diseases of mine and smelter workers. The author of this work was Aureolus Theophrastus Bombastus von Hohenheim, usually known as Paracelsus. The book, published posthumously, was entitled *Von der Bergsucht und anderen Bergkrankheiten*. Paracelsus was born in Switzerland at Einsiedeln near Schwyz. His father was a doctor, an able, well-educated man who had considerable experience of chemistry and metallurgy. In 1502 he was called to be town physician at Villach where the Tyrolese alchemist Sigismund Függer owned mines and maintained a mining school. From him the young Paracelsus received his first instruction in the extraction of drugs from plants and in the methods of identifying metals and producing chemical compounds.

Paracelsus studied medicine under Leoniceno in Ferrara. Like all other medical students of his day he read the Greek and Arab authors, the chief authority being Galen. Having obtained his medical degree in 1515, he wandered for many years all over Europe, enlisting in various armies and visiting numerous countries as far apart as England and Turkey. During his wanderings he came into contact with people in every walk of life, and

wherever he went he sought knowledge not only from the learned abbots and bishops but also from barbers, gypsies, midwives, executioners, and fortune-tellers. He learned a great deal about medical practice, and incidentally acquired an unusual knowledge of folk-medicine and a permanent taste for low company. He thought and spoke in the language of the people and was popular as no other physician before him. Finally returning to Strassburg in 1526 he began to practise medicine, but in 1527 he became town physician in Basel and lecturer at the university.

In the substance and manner of his teaching Paracelsus soon revealed his intention of reforming and rejuvenating medical theory and practice. Contrary to custom he held his lectures in German and not in Latin; he also wrote many of his books in his mother tongue. His lectures were very popular and soon became overcrowded. Imbued with a lifelong reverence for Hippocrates, implanted by his teachers in Italy, he began his campaign of reform by publicly burning the works of Galen and Ibn Sina (Avicenna). Instead of commenting upon the ancients, the customary manner of instruction at the time, he preferred to base his lectures on his own experiences. His popularity soon aroused the envy of his colleagues, and his attitude of derision and contempt for medical scholasticism added to his unpopularity with established authority. He had a truculent independent spirit and was one of the few writers who ever advanced medicine by quarrelling about it. Far in advance of his time, Paracelsus taught physicians to substitute chemical therapeutics for alchemy. He made opium, mercury, lead, sulphur, iron, arsenic, copper sulphate, and potassium sulphate part of the pharmacopoeia.

In 1528 he was forced to leave Basel, and the next year found him in Nuremberg, where he published the work in which he established the use of mercury in the treatment of syphilis. But the university authorities prohibited any further printing of his books and he set off again on his wanderings, visiting Scandinavia and Saxony. In 1536 he reached Augsburg and then Innsbruck, where there was an epidemic of bubonic plague. He practised all over Germany and Austria with varying success and in 1538 he lived at Klagenfurt in Carinthia. Two years later he was invited to work in Salzburg by Prince Ernst of Bavaria. His

hope of finding a haven of contentment was fulfilled but briefly, for in 1541 he met his end from a wound in a tavern brawl in Salzburg.

We have seen how Paracelsus while only a boy learned chemistry, metallurgy, pharmacy, and botany from his father in the mining centre of Villach. Soon after he qualified in medicine he was employed for five years in the smelting plant at Schwaz, in Tyrol. Later on, during his journeys through Hungary, Denmark, and Sweden, he learned about the mines of those countries. About 1533 he passed through the industrialized Inn valley which contained many mines, and it is likely that conditions in the mines and the diseases of miners awakened his interest then. But in 1537 the management of the Függer mines called him back to Villach to take charge of the metallurgical work there. It is evident that Paracelsus had ample opportunity to study the mining industry, to observe the diseases of miners, and to study the effects of various minerals and metals on the human organism.

In order to understand his views upon the toxicology of metals one must look to the theories held by alchemists long before this time; for example, the theory of the tartarus diseases was introduced into Europe from the Orient in the thirteenth century. Tartarus was a general term comprising all forms of precipitation or sedimentation. According to Paracelsus the tartarus itself was not a simple substance but a mixture of mercury, sulphur, and salt. We are not to think of these three substances as identical with those we know today, but rather as the three basic categories of matter. The names allude to the reactions of substances when exposed to heat; sulphur is that which burns, quicksilver that which evaporates, and salt that which resists heat. There are as many kinds of mercury, sulphur, and salt as there are substances.

He knew that work in certain mines gave rise to dyspnoea, cough, and even cachexia, and he thought that these symptoms were due to the climate or vapour of the mines. It is striking, however, that he refers to no protective apparatus such as the veils to be worn by miners mentioned by Agricola. Nor does Paracelsus pay any special attention to dust as a causative factor in the diseases of miners. Although he makes correct clinical observations, he then turns to weird alchemical theories to explain

them. For example, he strives hard to make the respiratory diseases of miners fit the theory of the tartarus.

> The lung sickness comes through the power of the stars, in that their peculiar characters are boiled out, which settle on the lungs in three different ways: in a mercurial manner like a sublimated smoke that coagulates, like a salt spirit, which passes from resolution to coagulation, and thirdly, like a sulphur, which is precipitated on the walls by roasting.

He goes on to identify this process with the slow deposition in a barrel of clear wine of a layer of wine-stone or tartarus.

In the second and third books of *Von der Bergsucht*, Paracelsus describes the diseases of smelter workers and metallurgists. The numerous correct observations that he made are evidence of his own experiences in the mines and metal refineries. He was well acquainted with the Tyrol, Carinthia, and Carniola regions where mercury was mined and refined. He recognized the poisonous effects of various metals and differentiated acute and chronic poisoning. In his detailed description of mercurialism, he mentions most of the important symptoms. Although his monograph was only a beginning, there can be no doubt it was an important one. His work is unique in the literature of the sixteenth century and it exerted a definite influence for at least 150 years after it was written.

The Father of Occupational Medicine

Exactly a hundred years after Paracelsus began his study of the toxicology of metals in the valley of the Inn there was born in Italy in 1633 a baby who was to become a great pioneer of the seventeenth century and the Father of Occupational Medicine. Ramazzini lived to the age of eighty-one. The first edition of the work to which he owes his immortality, *De Morbis Artificum Diatriba*, was published in 1700 when he was sixty-seven years of age. This book is to the history of occupational diseases what the *De Fabrica Humani Corporis* (1543) of Vesalius is to anatomy, Harvey's *De Motu Cordis* (1628) to physiology, and Morgagni's *De Sedibus et Causis Morborum* (1761) to pathology.

Ramazzini was born at Carpi near Modena. He studied phil-

osophy and medicine at the University of Parma, where he took his medical degree in 1659. After practising for twelve years in Rome and in Carpi, he was appointed in 1671 professor of medicine in the newly restored University of Modena. He worked there for thirty years, eighteen of which were spent at the University. He made accurate studies in epidemiology, describing the outbreak of lathyrism at Modena in 1690 as well as several outbreaks of malaria. No doubt he would be astonished could he know that his fame today is based not upon his *Constitutiones Epidemicae* but upon his work on occupational diseases. Equally he might be somewhat surprised to learn that, since 1907, his countrymen in Florence have published in his honour a journal of social hygiene known as *Il Ramazzini*.

In 1700 he accepted the call of the Venetian Senate to the chair of medicine in Padua and his fame spread rapidly throughout Italy and all over Europe. About 1703 he began to lose the sight of both eyes, presumably as a result of retinal arteriosclerosis. His grandsons helped him, especially with his reading and writing, but in 1707, at the age of seventy-four, he asked leave of Venice to retire. Instead he was promoted to the great distinction of being President of the Venetian College and told that he need not lecture if he felt indisposed. On his eighty-first birthday, as he was preparing in the afternoon to go to lecture to his students, he was seized with apoplexy and died within twelve hours. He was buried in the church of the Nuns at St Helena in Padua, but in an unknown grave.

From the writings of his nephew we know that he was lean and dark-complexioned, with black curly hair and attractive dark eyes. He was a hard worker and keen observer, paid little attention to the affairs of his household, and avoided games as one would a mad dog or a snake. He was active and walked so fast that his regular escort of students could hardly keep up with him. His hair became prematurely white, but was hidden by an elegant and becoming wig. He liked to dress well and suitably, but scorned doctors who were soft or self-seeking. When he had anything to do in public he was timid and nervous, especially at first. He eagerly cultivated friendships with the learned, and with so much affection and homage that they paid him honour at all

times. At work his chief asset was a capacity for exact observation which enabled him, in spite of the lack of knowledge of anatomy, physiology, chemistry, and bacteriology of his time, to arrive at surprisingly accurate conclusions. Unlike Paracelsus he was a singularly untravelled man, and there is no evidence that he ever left Italy or, indeed, ever went farther than Rome.

In the town of Modena where Ramazzini lived, the inhabitants of the tall, crowded houses, acting up to the best sanitary standards of the time, saw to it that the cesspits, which were connected with the drains that ran in different directions through the streets, should be cleaned out in each house once in every three years. And so Ramazzini wrote:

> On one occasion when that work was going forward in our house, I observed one of the labourers making extraordinary exertions to get through with his task. I pitied him on account of the cruel nature of the work and asked him why he toiled so feverishly and did not try to avoid exhaustion by working at a slower pace. Whereupon the poor fellow lifted his eyes up out of the pit, fixed them upon me, and said: 'No one who has not tried it can imagine what it costs to spend more than four hours in this place. It is as bad as going blind.'

The inquiry thus auspiciously begun bore a rich fruit. Ramazzini did not forget that cleaner of privies. He was profoundly convinced of the importance of the mechanical arts for the progress of civilization, but he was equally impressed by the wretched conditions of those engaged in these arts. He said he had to confess that many arts are the cause of grave injury to those who practise them, and that many an artisan has looked to his craft as a means to support life and raise a family, but all he has got from it is some deadly disease, with the result that he has departed this life cursing the craft to which he has applied himself.

> Medicine, like jurisprudence, should make a contribution to the well-being of workers and see to it that, so far as possible, they should exercise their callings without harm. So I for my part have done what I could and have not thought it unbecoming to make my way into the lowliest workshops and study the mysteries of the mechanic arts.

In the course of carrying out his resolve Ramazzini inquired into the conditions of work and the occupational diseases of the

following types of workers: miners of metals, healers by inunction, chemists, potters, tinsmiths, glass workers and mirror makers, painters, sulphur workers, blacksmiths, workers with gypsum and lime, apothecaries, cleaners of privies and cesspits, fullers, oil pressers, tanners, cheese makers and other workers at dirty trades, tobacco workers, corpse carriers, midwives, wet-nurses, vintners and brewers, bakers and millers, starch makers, sifters and measurers of grain, stone cutters, laundresses, workers who handle flax, hemp, and silk, bathmen, salt makers, workers who stand, sedentary workers, runners, grooms, porters, athletes, those who strain their eyes over fine work, voice trainers, singers, farmers, fishermen, soldiers, learned men, nuns, printers, scribes and notaries, confectioners, weavers, copper smiths, carpenters, grinders of razors and lancets, brick makers, well diggers, sailors and rowers, hunters, and soap makers.

Perhaps Ramazzini erred in attributing disease to trades which were merely offensive to the sense of smell; certainly he was wrong in imputing fever to limewashed walls, but he fully realized many things which would today come under the heading of welfare recommendations. For example, he counsels rest intervals in work of prolonged duration and dwells much on the need for exercise and change of posture, being convinced of the importance of faulty posture in producing ill health in many trades. He condemns want of ventilation and unsuitable temperatures and urges that workers in dusty trades should, in default of any known exhaust system, work in spacious rooms with their backs to the draught and should wash their faces, rinse out their mouths with water, and quit work in such trades immediately symptoms of respiratory disease show that the lungs are threatened.

As one result of his inquiries we find Ramazzini, among other wise counsels, making a striking addition to the Hippocratic art:

> When a doctor visits a working-class home he should be content to sit on a three-legged stool, if there isn't a gilded chair, and he should take time for his examination; and to the questions recommended by Hippocrates, he should add one more – What is your occupation?

Did ever a man announce with more point or with less fuss a

revolutionary innovation? In one innocent-sounding sentence we find that Ramazzini characterizes and supersedes the medical science and the medical practice of two thousand years. This simple piece of practical advice throws a strong light on our industrial civilization. In the main it is only when dealing with the common people that the doctor must think of the dangerous trades. Ramazzini made this his motto – *Medici munus plebeios curantis est interrogare quas artes exerceant.*

The Industrial Revolution

The complex series of events which changed the face of England between the years 1760 and 1830 has been aptly compared to a revolution. The term *die industrielle Revolution* was used as early as 1845 by Friedrich Engels in his *Die Lage der arbeitenden Klasse in England,* but the credit for originating the comparison is generally ascribed to Arnold Toynbee, whose book, unfinished when he died, was published in 1884 under the title *Lectures on the Industrial Revolution in England.*

Whether the name is appropriate has been much debated. Clearly the changes which took place were not merely industrial but also social and intellectual, and equally they did not occur with the suddenness of a revolution. But the great inventions which distinguished these years played so decisive a part in creating the new kind of society that the term industrial revolution is not too violent a description of the changes they produced. It is now an established phrase like the Renaissance, or the Middle Ages, accepted in common speech and having a well-understood meaning.

The Industrial Revolution in England was not a war, nor was it a revolt or anarchy; it was a stage in a long process of social evolution. The bounty of nature had provided water-power, coal, and iron; the great inventors had discovered or contrived new means of turning them to man's use; the philosophy of Jeremy Bentham and Adam Smith had furnished new inspiration to man's aspirations; and the rapidly increasing population had redistributed itself. Those were the main inherent factors, but concurrently American cotton had come to Lancashire. Some of

the inventors were poor operatives guided by mechanical faculty and ingenuity in their own industry. John Kay invented the weaver's flying shuttle about 1733, and in the next twenty years came Hargreaves' spinning-jenny, Arkwright's frame, and Crompton's mule for cotton spinning. Again, James Watt's condenser for the steam engine was exploited by Matthew Boulton in 1765, and within a few years steam became the dominant power in the staple industries of Britain.

The iron-working trades prospered under iron masters such as John Roebuck and Richard Crawshay. When coal took the place of charcoal in the smelting of iron the manufacturer could set up his plant in the neighbourhood of a coalfield. So the Black Country was created and enabled England to turn to account the good fortune which had given her an abundance of coal and iron conveniently placed near her ports. Thus iron and coal came at the advent of the new railway and transport systems, and, with cotton weaving, placed England at the head of industrial Europe. The canals of James Brindley and the roads of Thomas Telford and John Macadam facilitated inter-communication, pack-horses gave way to coaches and coaches to railways. In half a century the population of England rose from seven to twelve millions, redistributed from the south to the north, and from the land to the towns. Vast evil-smelling, jerry-built slums grew up overnight, and quick fortunes were made by the few out of the ill-paid toil of the many.

The hardships of the Industrial Revolution fell with particular severity on the women and children driven by two circumstances into the new factories. The women were enticed by inflated wages. The children were conscripted by the existing poor law apprenticeship. Both women and children were helpless to protect themselves against the demands and cupidity of the employers of cheap labour. The children from 5 to 12 years of age were carried off in wagon-loads from the rural districts to the factory towns, and made to work for extremely long hours under cruel and unhealthy conditions. William Cobbett accused England of building her industrial dominance on the labour of 30,000 little girls. As the eighteenth century came to a close there was a gradually awakening interest in the sad plight of these children,

and this led to the passing of the first factory act, the *Health and Morals of Apprentices Act, 1802*. The employers urged that the longer the hours, the greater their profits, that the industrial system was absolutely necessary to national supremacy and security and that upon its success depended the national life and credit. The men workers complained that their individual health, livelihood, and well-being, and that of their womenfolk and children, were being sacrificed to the long hours and evil conditions of the mills.

In the second *Factory Act, 1819*, a few concessions were made. It stipulated nine years as the minimum age for the employment of children and it limited their working hours, but it did not apply to all textile factories. But one important principle it did establish. Parliament, in the teeth of violent opposition, had won the right to extend the law to workers other than bound apprentices, and as the opponents of the Bill so rightly judged, this was indeed the thin end of the wedge. Though the Bill was badly mutilated, the weak and feeble Act which emerged was to become the Magna Carta of childhood; thereafter the protection of the children of the poor first from toil and then from bodily starvation and ignorance began.

The *Factory Act, 1819*, served to bring the question of the mills before the public eye, and there was an increasing demand for investigation and control of the many abuses which were becoming evident. The first moves came not from the workers themselves, which is not surprising in view of the almost universal illiteracy of the labouring classes, but from public-spirited men who, wishing to establish good conditions in their own factories, found themselves placed at a disadvantage by the cut-throat practices of their competitors. The demand for legislation to restrict the exploitation of child labour was made first by them, and the popular clamour and widespread agitation which characterized subsequent Bills was not really canalized into an effective weapon until the birth of the *Ten Hours Movement*, which sought to reduce the hours of all workers – men, women, and children alike. The struggle for the Ten Hours Bill was long and cruel, and lasted from 1830 to 1847. In its later years groups of ragged children went about the towns chanting:

We will have the Ten Hours Bill;
Yes we will, yes we will.

Robert Owen, himself a cotton manufacturer, was one of the early heroes of this conflict, the first of the long line of humanitarians, who fought for the education and health of the children entering his mills at New Lanark. Both Owen and Pitt were converted to free trade by the work of Adam Smith, the professor-economist of Glasgow, who moved the industrial world in 1776 by his great book *Enquiry into the Nature and Causes of the Wealth of Nations*, in which he holds that labour, being the source of national wealth, must have freedom to pursue its own course of interest. Owen was taught his factory principles by his business partner, Jeremy Bentham, the father of English law reform, who in 1789 wrote his *Introduction to Principles of Morals and Legislation.* In this book he inspired the whole world with his doctrine of utility, the aim of which was to prevent mischief, pain, evil, or unhappiness. He claimed that the object of all legislation must be the greatest happiness of the greatest number.

Dr Thomas Percival of Manchester initiated the first medical services on behalf of the industrial worker, by his voluntary Board of Health, in 1796. As educationalist, mathematician, and pioneer, he was the friend of Voltaire and Condorcet, and the leader of that long line of doctors who saved the health of the factory worker. It was Robert Owen and Thomas Percival who brought Michael Sadler into the reform movement, and he in his turn inspired Anthony Ashley Cooper, at first Viscount Ashley and afterwards the seventh Earl of Shaftesbury, a life-long apostle and exponent of practical altruism who never ceased to fight for reform.

His contemporaries in the House of Lords, some of whom owned the mills, the mines, and the land, remained for a long time apathetic and deaf to his appeals. The ruling doctrine of the time was that progress was measured by trade and profits, that popular misery could be cured only by encouraging capital; that the needs of the new industrial system must govern and limit the development of social life. No mill owner thought he could afford fair wages, especially as new machinery was paid for out of profits. The wages of industrial workers were forced down lower

and lower, longer hours were worked, and many went hungry. Religion and money making had contrived to get on the same side, and it was possible for men at one and the same time to be very rich, very greedy, very cruel, and very orthodox. So long as the factory owners professed to fear God, continued to hold morning prayers with their families, and attended church or chapel on Sunday they considered their duty to their fellow men well done.

In combating this situation, men like Percival and Shaftesbury introduced many reforms, but their chief contribution was a spirit of humanitarianism and an understanding of the health and social requirements of the worker. Nor were all the employers flint-hearted. David Dale and Robert Owen set the classical example of beneficent autocracy in industry, and men like Josiah Wedgwood, Matthew Boulton, and John Wilkinson maintained a high standard in their treatment of workers and in their business methods.

Neither was Robert Owen the only employer who considered the interests of the child worker. At the apprentice houses run by Samuel Oldknow, a cotton spinner at Mellor, and by the Gregs at Styal, children were well fed, well housed and taught, and kindly treated. In such factories, men like these sowed the seeds of the future State education of children and of the regulation of child labour. Indeed we can say that the *Factory Act, 1833*, supported by these reformers, fortuitously drove the public of Great Britain to accept education, universal and compulsory.

The Birth of Industrial Medicine in England

Whereas, through Ramazzini, the idea of asking a patient what is his occupation had birth in Italy, industrial medicine itself was born in England. Because the Industrial Revolution began in England it naturally came about that English doctors were the first to assume responsibility for the health of the worker in industry. The special task of investigating the health hazards in various occupations began early in the nineteenth century in the West Riding of Yorkshire and in Lancashire, when Charles

Turner Thackrah, a clinician who practised in Leeds, decided to devote his life to preventive medicine of a new kind.

The manufacturing activities of Leeds at that time, as today, were very varied, and at least 128 trades and occupations existed there. The reason for such a diversity of manufactures was, as the Hammonds have pointed out, that Leeds, like certain other northern cities, was not the creation of the Industrial Revolution: it was a town of antiquity already renowned as the centre of special industries and trades. Such a town was not thrown up by the Industrial Revolution, but rather overwhelmed by it; its population increased from 60,000 to 130,000 between 1801 and 1831. Thackrah expounded his work in a short book of 220 pages published in 1831 with the title, *The Effects of the Principal Arts, Trades and Professions, and of Civic States and Habits of Living, on Health and Longevity, with Suggestions for the Removal of many of the Agents which produce Disease and shorten the Duration of Life*. It was the first treatise of any kind to be written in England on this subject. For comprehensiveness, first-hand clinical experience, and constructive proposals for improvements, Thackrah's monograph is superior to that of Ramazzini. It attracted attention from both medical men and laymen at the time that it appeared, and there is no doubt that it played an important part in stimulating the factory and health legislation which mitigated some of the worst features of the Industrial Revolution.

Thackrah was born in Yorkshire in 1795 and became apprenticed to a prominent local practitioner. Like the more ambitious of his contemporaries he came to London, then the only serious centre for medical education in England, to complete his training in one of the larger hospitals. At that time the clinical staff at Guy's Hospital was the most active centre of research in London, and through their publications they were gaining both a national and an international reputation for their hospital. Sir Astley Cooper, his immediate teacher and inspirer of much of his work, was at the height of his powers and Thackrah remained in this stimulating atmosphere for several years as one of his assistants. During these years Bright, Addison, and Hodgkin, who had all been attracted to Guy's shortly after their graduation at Edinburgh,

were his contemporaries. After leaving Guy's, Thackrah returned to Yorkshire and settled in practice at Leeds. In 1826 he opened the Leeds School of Anatomy, which soon became so successful that it grew into the complete medical school from which the present Leeds University School of Medicine has developed. Thackrah may thus be regarded as one of the founders of provincial medical education in England, and as one of the vigorous and successful opponents of the claims to monopoly which had long been maintained by the two Royal Colleges in London.

What directed Thackrah's interest to industrial diseases is not clearly known. It is possible that it resulted from some personal contact with Robert Owen, who visited Leeds during Thackrah's apprenticeship to study the methods of working of the local factories and the condition of their employees, and the two men exchanged views at the time. Most probably Thackrah's interest in the medical aspects of the problem were the combined result both of his own professional activities amongst the poorer classes in Leeds and of Owen's forceful representation of the urgency of reform. Whatever might have been the impetus, the results of his activities materialized in a small volume which appeared in 1830, and which was soon followed by a much larger and definitive second edition in 1831, a year before his death. His own intentions in publishing the book may be learned from the two following paragraphs:

> The object of this paper is to excite the public attention to the subject. Myself and my pupils have personally and carefully inspected the state of the artisans in most kinds of manufacture, examined the agencies believed to be injurious, conversed on the subjects with masters, overlookers, and the more intelligent workmen, and obtained many tables illustrating the characters of the disorders prevalent in the several kinds of employ. It will be remembered that the subject is new. I have had therefore to enter a new track without guide or assistance.
>
> Most persons, who reflect on the subject, will be inclined to admit that our employments are in a considerable degree injurious to health: but they believe, or profess to believe, that the evils can produce only pain and discontent. From a reference to fact and observations I reply, that in many of our occupations, the injurious agents might be immediately removed or diminished. Evils are suffered to exist, even when the

means of correction are known and easily applied. Thoughtlessness or apathy is the only obstacle to success.

The book is divided into four sections, which deal successively with operatives, dealers or shop-keepers, merchants and master manufacturers, and professional men. Much the longest and most interesting section deals with the operatives. Before proceeding to diseases associated with particular trades, two of the more general of Thackrah's comments deserve attention: the employment of children, and the frequent references to bad occupational postures as sources of disability. The severity of the conditions of child labour, especially in cotton mills, had been lessened at the time Thackrah wrote, but that they had been far from eliminated can be seen from his comments upon work in the flax mills.

> Children from seven to fifteen years of age go to work at half past five in the morning, and leave at seven in the evening, or at half past six and leave at eight, and thus spend twelve hours a day, for five or six years, in an atmosphere of flax dust. Serious injury from such employment we should expect at any age, but especially during the period of growth. The employment of young children in any labour is wrong. The term of physical growth ought not to be a term of physical exertion. Light and varied motions should be the only effort, motions excited by the will, not by the taskmaster, the run and the leap of an unshackled spirit. How different is the scene in a manufacturing district! No man of humanity can reflect without distress on the state of thousands of children, many from six to seven years of age, roused from their beds at an early hour, hurried to the mills, and kept there, with an interval of only forty minutes, till a late hour at night: kept, moreover, in an atmosphere impure, not only as the air of a town, not only defective in ventilation, but as loaded also with noxious dust. Health! Cleanliness! Mental improvement! How are they regarded? Recreation is out of the question. There is scarcely time for meals. The very period of sleep, so necessary for the young, is too often abridged. Nay, children are sometimes worked even in the night!

Bad posture at work, though by no means confined to the textile trade and its various branches, was particularly prevalent amongst persons following such occupations. It was probably the large numbers of such tradesmen in Leeds in his time that drew Thackrah's attention the more forcibly to the disabilities

and deformities that resulted from it. Weavers, burlers, cloth-drawers, and especially tailors are remarked upon unfavourably.

> We see no plump and rosy tailors: none of fine form and strong muscle. The spine is generally curved. Pulmonary consumption is also frequent. Let a hole be made in a board of the circumference of the tailor's body, and let his seat be placed below it. The eyes and the hands will then be sufficiently near his work: his spine will not be unnaturally bent, and his chest and abdomen will be free.

Thackrah wrote of the dust diseases of the lungs affecting miners and grinders of metals.

> In the mines in the North of England, the workmen, I am informed, suffer considerably when employed in ore in the sandstone, but are sensible of no inconvenience where the ore is in limestone. I am indebted to an intelligent friend for the following information: The reason they assign is, that the latter is full of vertical and other fissures, which allow the superincumbent beds of water to percolate through the roof of the mine: whilst the sandstone strata, which are impervious to water, preserve the mine quite dry: consequently, the minute particles of rock formed by blasting or the pickaxe are kept in a dry state within the sandstone mine, forming, as it were, an atmosphere of dust, which the miner is constantly inhaling. Miners rarely work for more than six hours a day, yet they seldom attain the age of 40. A parallel case to that of the miners occurs in the grinders of Sheffield. Dr Knight, in the *North of England Medical Journal*, states that the fork-grinders, who use a dry grindstone, die at the age of 28 or 32, while the table-knife grinders, who work on wet stones, survive to between 40 and 50.

In both trades, mining and grinding, Thackrah comments on the common association of dust inhalation and tuberculosis. Amongst the men filing iron castings to remove burnt-on sand, pulmonary diseases were prevalent. To protect such workmen he recommends, in addition to proper ventilation, the introduction of suitable magnetic mouthpieces.

Amongst other specific industrial diseases discussed by Thackrah, perhaps the most important were those associated with chronic lead poisoning amongst house-painters and the potters making glazed ware. Such men were frequently attacked with colic, suffered from constipation, and ultimately developed lead palsy. Thackrah made specific recommendations for the elimination of lead poisoning as a trade disease amongst pottery workers.

Could not the process be effected without the immersion of the hands in the metallic solution? Or could it not be effected by a machine? Or could not some article less noxious be substituted for the lead? I am told, indeed, by an intelligent manufacturer of earthenware in Leeds, that the comparative cheapness of the leaden glaze is the chief recommendation. Surely humanity forbids that the health of workmen, and that of the poor at large, should be sacrificed to the saving of halfpence in the price of pots. The total disuse of lead in glaze is highly desirable.

Thackrah's work in the field of occupational hygiene has never failed of recognition. He was undoubtedly an able and energetic man with a strong social sense, vigorous, pertinacious, and courageous, and was determined that his findings should be made widely known at a time when the advocacy of reforms was even less popular than usual. The publication of such a volume from a member of the medical profession, with the prestige that special technical knowledge gave him, considerably aided the parliamentary advocates for the limitation of factory abuses.

Sir John Simon, the first Chief Medical Officer to the Local Government Board (now the Ministry of Health), referred to Thackrah's work as being comparable as a contribution to preventive medicine with the work of Jenner on smallpox. He wrote:

This special service of investigating health in various branches of industry, Thackrah set himself to render: not under any official obligation or inducement, nor with any subvention from Government, but as his own free gift to a public cause. Not less meritorious than the assiduity and the care for truth with which he collected his facts were the unprejudiced good sense and moderation with which he weighed them; and the service thus rendered by Thackrah deserves grateful recognition. By his eminently trustworthy book, he, more than fifty years ago, made it a matter of common knowledge, and of State responsibility, that, with certain of our chief industries, special influences, often of an evidently removable kind, are apt to be associated, which, if permitted to remain, give painful disease and premature disablement or death of the employed persons.

Thackrah's crowded life was brought to an untimely end by his death from tuberculosis in 1833 at the age of thirty-seven.

The Factory Inspectorate

The Factory Act, 1833, was the first really effective legislative measure in the industrial field. It was entitled *An Act to Regulate the Labour of Children in the Mills and Factories of the United Kingdom*. It applied to all textile factories where steam or water power was used, including flax, hemp, and silk. It forbade night work for those under eighteen and restricted their hours to twelve a day and sixty-nine a week; factory schools were established and all children under the age of thirteen were required to attend for at least two hours a day. The minimum age was set at nine. Prior to the passing of this Act the age of a child was established by a certificate, often very dubious, from the parent; but by the Act of 1833 a certificate of age was required from a medical man to the effect that a child was of the ordinary strength and appearance of a child exceeding nine years or other specified age.

But the most important provision of all was the establishment of a Factory Inspectorate to administer the Act. The *Factory Acts of 1819* and *1825* had left the voluntary local system of factory inspection by visitors unchanged, but now the principle of enforcement of the law by means of statutory inspection was introduced and the first four inspectors were appointed. They were Leonard Horner, Thomas J. Howell, Robert Rickards, and Robert J. Saunders. These men were given the power of entry into all factories covered by the Act, power of prosecution of recalcitrant owners and, in particular, responsibility for setting up and inspecting factory schools. The country was fortunate in that the early holders of office were men of great moral courage and a wide experience of affairs, and the importance of their contributions to later reform cannot be over-emphasized. Indeed, it would be difficult to point to a single legislative measure of importance relating to the health of factory workers from that day to this which has not been initiated in whole or in part by the Factory Department.

Factory inspectors were charged with the enforcement of factory laws and had in addition the duty of persuading the management and the workers in factories to reduce nuisances. A

very important task of the inspectors was to bring about a further development of new laws by studying the actual working conditions and their effects on health.

A factory inspector must be a person of integrity who is by character well able to stand up to the employer. To become an inspector a man or woman must be either a graduate of a university or must hold the qualification of a recognized school of engineering. A candidate must pass oral and written examinations, attend lectures, and work for some weeks with an older inspector. After two years on probation, the candidate is required to pass an examination on factory law and the sanitary sciences before being appointed. Inspectors must treat as confidential all information concerning installations in factories and may discuss them outside the factory only in official reports and at legal prosecutions. Relations between inspectors, management, and workers have improved during the course of the years. At first factory inspectors reported much resistance from managements, many attempts at deception, and also disagreeable incidents. That this occurs even today is shown by the fact that in Great Britain before the Second World War more than 500 firms were penalized yearly for over 1200 offences. But this represents a big improvement over the opening years of the twentieth century, when there were from 3000 to 4000 offences every year. The factory inspector of today has been aptly described as a friendly adviser, whose advice is backed by the knowledge that he has the power to enforce his recommendations.

The *Factories and Workshops Act, 1878*, created a centralized system of factory inspection with a Chief Inspector in London. The first man to hold this office was Alexander Redgrave. In 1884, there were, in addition to this Chief Inspector in London, five superintending inspectors, thirty inspectors, and ten juniors distributed throughout the country. In 1893 the first woman was appointed. It was not until 1896 that a medical man, Arthur Whitelegge, Medical Officer of Health for the West Riding of Yorkshire, who became Sir Arthur Whitelegge, was enlisted for the post of Chief Inspector, and, although he did not so act in a medical capacity, it was no doubt as a result of his advice that the post of Medical Inspector was created in 1898,

and Thomas Morison Legge, who later became Sir Thomas Legge, was appointed. In 1902 the first electrical inspector was appointed, and in 1903 the first inspector for dangerous trades. Each of these individual appointments has since been expanded into a body of several inspectors. In 1910 the authorized staff of inspectors and assistants consisted of 200 persons, in 1939 of 320, in 1944 of 440 persons. In that year 317,040 inspection visits and 63,852 other official visits were made to factories.

Thomas Morison Legge, 1863–1932

Born in Hong Kong, Legge came as a boy to England, where his father, Dr James Legge, was appointed Professor of Chinese in Oxford. The boy attended Magdalen College School, went to Bonn to learn German, and then joined Trinity College, Oxford, as a commoner. He completed his medical education at St Bartholomew's Hospital, graduated B.M., B.Ch. in 1890, and obtained the D.M. in 1894. Wishing to study preventive medicine, he paid a series of visits to the capitals of France, Germany, Belgium, Sweden, Norway, and Denmark, and wrote his first book, *Public Health in European Capitals*, in 1896. After working under Arthur Newsholme, Medical Officer of Health for Brighton, he was appointed in 1898 the first Medical Inspector of Factories and began what may be described as his life's work.

In the early part of his career in the Factory Department of the Home Office he was chiefly engaged in studying and dealing with the prevention of lead poisoning, the disease with which, more than any other, his name will always be associated. In 1912, with Dr K. W. Goadby, he wrote a book entitled *Lead Poisoning and Lead Absorption*. In subsequent years he investigated anthrax, glass-blowers' cataract, industrial cancer of the skin, toxic jaundice, and poisoning by phosphorus, arsenic, and mercury. His vast knowledge of his subject was recognized both at home and abroad; indeed, it is difficult to think of an industrial disease on which he could not speak with authority and experience. He received the C.B.E. in 1924 and a knighthood in 1925. He laid stress on the need for better education in occupational medicine as part of the curriculum in medical schools, and was untiring

in giving lectures to medical students in many hospitals. As a lecturer he showed a good sense of proportion as well as a sense of humour. He often punctuated his subject with historical anecdotes of great interest.

His love of foreign languages facilitated his studies of the history of his subject, and his knowledge of German gave us his excellent translation of Rambousek's *Industrial Poisoning from Fumes, Gases, and Poisons of Manufacturing Processes.* There was a strong artistic side to his nature, strengthened by his upbringing in Oxford in the atmosphere of William Morris (1834–96), Dante Gabriel Rossetti, and Edward Burne-Jones. He admired the work of these men, who protested against the ugliness of Victorian commercialism, asserting the necessity for natural decoration and pure colour in chintzes, carpets, metalwork, and stained glass. The *Kelmscott Chaucer*, decidedly the crowning glory of Morris's printing-press, was finished in June 1896 after five years' planning. Legge also admired the work of the Belgian sculptor of labour, Constantin Meunier, and his artistic eye sought and found much beauty in industry. He was never happier than when in his spare time after a day's work he was making a water-colour drawing of slag-heaps, cranes on the Clyde, or kilns at Hanley. But his chief hobby was the study and collection of medieval stained glass, and he was an accepted authority on Trade Guild windows.

In 1921 Legge went to Geneva as representative of the British Government at the International Labour Conference organized by M. Albert Thomas. He there helped to draft the original White-lead Paint Convention which prohibited its use for the internal painting of buildings. In 1926, taking the view that Regulations for the inside painting of buildings should be given a fair trial before adopting the drastic remedy of prohibition, the British Government refused to ratify the Geneva White-lead Paint Convention. Believing the drastic method to be indispensable, Legge felt that he could not bring himself to administer a method of prevention from which he could not foresee satisfactory results. He therefore resigned his appointment as H.M. Senior Medical Inspector of Factories. The fact that he was a Civil Servant and could in no sense be responsible for any policy which a higher

authority deemed advisable did not appeal to him on this occasion. In retirement he felt keenly the loss of contact with both employer and worker in the factory.

It was therefore with real pleasure that he accepted in 1929 the post of Medical Adviser to the Trades Union Congress. In the three years which followed he wrote his book on *Industrial Maladies*, and when he realized that his death might befall before the book was ready for the press, left directions that his colleague, Dr S. A. Henry, should edit the work. And so passed a great Englishman, one of the finest of our Civil Servants, a man who by his energy, intellectual integrity, and common sense achieved more for the protection of the worker against injury and disease than anybody else in all history. His life work lives on, to the greatest benefit of the worker in industry throughout the world.

CHAPTER THREE

LEGISLATION AFFECTING THE WORKER

The Factories Acts of 1937 and 1948 – Notification of Industrial Diseases – The Disabled Persons (Employment) Act, 1944 – Human Relationships in Industry – The National Insurance (Industrial Injuries) Act, 1946 – Prescribed Industrial Diseases

THIS chapter gives an outline of the legislation in force today which protects the worker from disease and accident, pays him benefit, and helps him towards rehabilitation when he is sick or injured. In addition to what is described here there are of course special regulations for particular trades and processes which would fill many volumes the size of the present one.

THE FACTORIES ACTS OF 1937 AND 1948

In addition to consolidating the previous Acts, the *Factories Act, 1937*, as amended by the brief Act of 1948, strengthened the provisions for safety and health and the requirements for first-aid installations. Important safety measures include regulations for the construction of machinery. These specify that every set-screw and bolt on a revolving shaft shall be sunk and that gearing shall be encased. Any person selling or renting a machine that does not comply with the requirements of the law shall be fined. If illnesses occurring in a factory are traceable to the nature of a particular job or if by reason of changes in any process the risk of injury arises, the Secretary of State is authorized to require arrangements for the medical supervision of the endangered persons. He also is authorized to prescribe the maximum weights to be lifted, carried, or moved by any class of person, and to require the attending physician to report all industrial diseases, not just those enumerated in earlier laws.

Factory Orders, 1944, published by the Ministry of Labour and National Service, is a book of 388 pages. Under general provisions

it includes five on health (cleanliness, lighting); eight on safety (hoists, chains, and cranes); and three on welfare (first-aid). There are also twenty-two welfare regulations for particular trades (cement works, bakeries, laundries, sugar factories, and so on), and forty-eight for safety and health in particular trades. Other regulations concern lead paint, the employment of women and young persons in lead processes, notification and investigation of accidents and industrial diseases, and homework. These rules and regulations show not only the tendency to make provisions against all the important causes of accidents and industrial diseases but, even more, the tendency to adapt all regulations to the special peculiarities of the work and to make the provisions as clear and definite as possible.

Notification of Industrial Diseases

From 1878 until 1940 provisions for the control of dangerous trades were made by the Home Office, and subsequently by the Ministry of Labour and National Service. Early knowledge of certain industrial diseases was obtained by placing an obligation on the medical practitioner to notify the Chief Inspector of Factories, and on the employer to notify both the district inspector of factories and the certifying surgeon, now the *appointed factory doctor*. Since 1896 an increasing number of diseases has been made notifiable. Under the *Factories Act, 1948*, the Chief Inspector of Factories must be informed of poisoning arising under fourteen headings:

Lead poisoning	Chronic benzene poisoning
Phosphorus poisoning	Compressed-air illness
Manganese poisoning	Anthrax
Arsenical poisoning	Toxic jaundice
Mercurial poisoning	Toxic anaemia
Carbon bisulphide poisoning	Epitheliomatous ulceration or
Aniline poisoning	Chrome ulceration

By the *Lead Paint (Protection against Poisoning) Act, 1926*, the practitioner must notify any case of lead poisoning contracted in painting any building. The aims of the *Factories Act, 1948* and of

the *National Insurance* (*Industrial Injuries*) *Act, 1946*, are, of course, quite different. The former is concerned with the prevention and control of occupational accidents and diseases, and the latter with those workers affected by occupational accidents or prescribed diseases. Notification is an important ancillary in the prevention and control of occupational accidents and of certain occupational diseases. Thus we find that the list of diseases prescribed under the *Industrial Injuries Act* is longer than that of the diseases required to be notified under the Factories Act, and in some cases they are described differently.

The Second World War saw the compulsory establishment of personnel services in certain prescribed factories (*Factories* (*Medical and Welfare Services*) *Order, 1940* and *Factories* (*Canteens*) *Orders, 1942 and 1943*). It is probable that the future will see industrial legislation focused on the individual rather than on a particular section of industry. There will also be a shift of emphasis from toxic and accident hazards to the personal needs of workers, both as individuals and as members of a group. But it is well to remember that only by the strictest measures of control, sometimes enforced by law, have industrial diseases and injuries due to accidents been reduced in the past, and regulations will still be needed, not so much to protect the worker from his employer, as to help the latter, who cannot be expected to know all the details of the numerous hazardous processes to be found in modern industry.

The Disabled Persons (Employment) Act, 1944

The shortage of labour during the Second World War became so acute that in 1941 a committee known as the *Interdepartmental Committee on the Rehabilitation and Resettlement of Disabled Persons* was appointed under the chairmanship of Mr George Tomlinson, M.P. The recommendations of the *Tomlinson Report* (*1942*) were set before Parliament and were accepted almost unaltered in a new Act known as the *Disabled Persons* (*Employment*) *Act, 1944*, in which the nation accepted the principle that any disability, medical or surgical, constitutes a claim upon the State

for assistance, both financial and by way of treatment and rehabilitation, so that the disabled person can be reinstated, when possible, into useful work. The Act defines a disabled person as one who –

> on account of injury, disease, or congenital deformity is substantially handicapped in obtaining or keeping employment, or in undertaking work of a kind which apart from that injury, disease, or deformity would be suited to his age, experience, and qualifications.

Such persons are entitled to apply for registration; and if accepted as disabled, they are assisted in various ways by the Ministry of Labour and National Service to obtain suitable work.

Since September 1945 a register of disabled persons has been maintained at all local offices of the Ministry of Labour. Registration is voluntary and application is made by disabled persons. To be accepted the following obligations have to be fulfilled: (*a*) The worker's disability must come within the definition laid down, (*b*) the disablement be likely to last for at least six months, and (*c*) the applicant must be ordinarily resident in Great Britain. Medical evidence may be necessary before an application is accepted, but rejection can be decided on only by the Disablement Advisory Committee which has the assistance of medical advice. Registration may be for one to five years and may be renewed. A person may be removed from the register if he fails to satisfy the definition, or if there is persistent and unreasonable refusal to take up suitable work, or if there is refusal without reasonable cause to complete a course of vocational training. Employers are required to engage a proportion of registered disabled persons; this usually amounts to 3 per cent when twenty or more men are employed. Certain industries, for example fishing fleets, are required to employ 0.1 per cent only of disabled persons.

There are some 800,000 names on the register, about half of these disabled persons being surgical cases. The medical group numbers just under one-third, and a group of patients with psychiatric symptoms makes up about 5 per cent of the total. The work of lift attendants and car park attendants is known as *designated employment*, and these jobs can be filled only by men who are on the register of disabled persons. The work of the

Ministry is carried out by local officers known as *Disablement Resettlement Officers* (D.R.O.), who maintain a close co-operation with hospitals, training institutions, and local industries. When necessary, patients are visited in hospital or at home and advised as to the proper steps to be taken to secure treatment, training, and placement.

The Ministry has set up training centres at which disabled persons are tested for vocational aptitude and instructed in suitable trades. Those so severely handicapped as to be unable for a considerable period, or permanently, to fit into an ordinary industrial environment may be accommodated in *sheltered workshops* under the auspices of the Ministry; such *remploy factories* have been set up in or near the main centres of population and provide for work to be done under specially favourable conditions. All the facilities provided at the industrial rehabilitation centres of the Ministry of Labour are free of cost to the trainee, who is paid his travelling expenses and maintenance allowances for his dependents. The training of disabled persons in Government Training Centres is carried out, where possible, side by side with that of the able bodied, so that the former may learn to work together with the latter. Those so seriously handicapped as to require a period of training of six months or more may be sent to one of the residential centres specially equipped for dealing with such cases, such as Queen Elizabeth's Training College for the Disabled, Leatherhead; St Loyes College, Exeter; or the Sir John Priestman Hospital, Finchale Abbey, near Durham, all of which are run by private voluntary organizations.

Rehabilitation should be based as far as possible on economic considerations, so that the individual obtains at the earliest possible opportunity both work and reward. Hospital services deal in the main with anatomical and functional recovery. With good hospital treatment the need for special measures of rehabilitation decreases, so that on economic grounds industry is fundamentally interested in future hospital policy. Many hospitals have ventured into the social field by changing the outlook and work of the almoner, but closer contacts with industry are still required.

The policy of setting up social service departments in hospitals is gradually providing an important link with industry; the func-

tion of the almoner is changing, and her increasing contact with managements, on behalf of the sick and injured worker, will be a real advance in hospital service. Industry itself frequently needs contact with hospitals; it must have knowledge, for instance, of the progress of key men under treatment, or it may wish to offer special facilities to an injured man to assist his recovery; this can best be done through the social service department of the hospital. Hospitals should consider the possibility of appointing their own industrial liaison officers, persons with practical experience of factory work, who would not only provide a link with the patient's employer but would advise the physician or surgeon concerning the physical and mental requirements of different occupations.

In addition to efficient hospital treatment and close contact between the almoner's department and industry, the co-operation of the employer is necessary for full resettlement. The promise of continued employment can remove the fear of insecurity, which is a potent means of retarding recovery. Many disabled workers already in employment are unwilling to put their names on the register of disabled persons for fear of losing their jobs, and because of a natural dislike of being classified as disabled.

A promise of alternative work can be made if the employee is unfit for his previous job, but, to be effective, this must be true alternative work under medical supervision and not light work specially created as a charity. The possibility of retraining for alternative work should be considered. This can be done only in the larger firms where such jobs are available. Government Training Centres and certain extra-industrial centres can, theoretically, cover the remainder of industry. It is important that employers should appreciate the fact that a man cannot be unfit one day and on the next, in a different environment, be really fit for full work. The goodwill of managers and foremen, who are the real executives of industry, must therefore be obtained for the worker during what is often an awkward phase in recovery – that period of time between being signed off by the insurance practitioner of the hospital and his return to his original occupation.

It is a primary responsibility of personnel management to play a part in the supervision of the return of the sick or injured worker to work. In the larger firms, personnel management and medical

services work in close collaboration. In the small industrial unit – for example, the factory with fewer than 300–400 employees – there is usually no special official appointed as personnel or welfare officer, and supervision therefore becomes the direct responsibility of the works manager. Because the majority of firms come within the small category, and because over 50 per cent of the factory workers are employed in units of 250 or fewer, the education of works managers and foremen in the meaning of rehabilitation and the part they must play in it becomes increasingly important.

Whereas firms may appreciate the need for *light or alternative work*, the provision of such work is difficult in industry as a whole and in the small organization it is frequently impossible. In larger firms a number of jobs may be especially allocated for convalescent sick and injured patients as part of general welfare schemes. The main criticism of this is that patients are often under inadequate medical supervision, and that this allocation may become no more than a method of dumping or losing unwanted workers for so-called philanthropic motives. Objections to alternative or light work can be summed up as follows. It is frequently hit-or-miss therapy; it is difficult to find in the majority of industries; it is difficult to keep under medical control and may retard functional recovery; it is frequently disliked by managers, foremen, and by workers themselves, for the productive effort of the industrial unit – the gang or team – may be hampered by the presence of a semi-fit man; managers and foremen may resent the increase in overhead expenses; and the patient is apt to be permanently lost sight of in the works.

In firms in which an industrial medical officer is employed, supervision of the return of the injured or sick worker to his job is one of his primary duties and is a main reason for further extension of occupational health services. Among other things he can obtain accurate information of the incidence of sickness and accidents and of the progress of sick and injured workers; he can review progress with hospital staffs and with medical practitioners. He should regard the local hospital as his base and make close contacts with it, for health departments in industry should regard themselves in certain respects as outposts of hospitals and should not work in isolation. He can develop an adequate follow-up

service, and here the help of a good nurse is of much importance; and he can build up a library of job references with a view to more effective placement.

The whole-time industrial medical officer should investigate the possibility of setting up, in his own organization, a *special rehabilitation workshop* for selected sick and injured workers. Selection could be made in collaboration with hospital physicians and surgeons, who might act in a consultative capacity in this respect and so themselves learn something of industrial conditions. The potential medical contribution to industrial rehabilitation is thus great, particularly in larger firms. This type of workshop, within industry itself, provides one good answer to the problem of rehabilitation, but it should essentially develop as a part of an occupational health service. The Austin and Vauxhall experiments are examples of this.

Employees other than those disabled by accident are admitted, for example, medical cases after prolonged absence or those individuals whose working capacity is so lowered by age or by slowly developing debility that they cannot continue their normal occupation. In a new environment, physical and mental capacity is reassessed under gentle conditions but, at the same time, a living wage is paid, and the man is eventually employed again in the factory proper. As soon as workers realize that an alternative to either full work or unemployment is available, they are relieved of much of the anxiety which results from economic insecurity. The worker has a double incentive, (*a*) to earn more than he would on compensation and so would be attracted to work in the shop, and (*b*) to strive towards his pre-disability rate. The man is employed in the shop at the sole discretion of the medical officer, and he returns eventually to his previous work, or to other full work for which he is suitably trained, in other parts of the factory.

A follow-up scheme operates in collaboration with shop superintendents. Such a scheme realizes the psychological needs of any incapacitated or handicapped person. It takes him away, early in his treatment, from the hospital atmosphere in which disability and disease are inevitably stressed. It removes financial anxiety and the fear of not being employed again. It alters a man's

attitude to his mates, to his manager, to the firm, and to his home, and the change is inevitably for the better, because he lacks reason for grievance. There is less tendency to retain the memory of disabilities. Morale is good and neurotic manifestations and recurrences are minimal. The scheme allows of a day-to-day contact between the medical staff and a section of semi-fit and disabled employees at work. Once the man is told, in simple language, of the immediate and ultimate effects of his disablement, and is reassured about his future employment, he is willing to cease worrying about it, and he transfers his load of anxiety to the shoulders of the doctor.

It is common knowledge that much time is lost in factories because of *neurotic dissatisfaction.* Doubts as to social security, boredom from the monotony of mechanized jobs, fear of unemployment or of inability to work all have a decidedly injurious psychological effect on the workman. In spite of the various efforts made to interest him in his task, including social work of all kinds, incentive schemes, sharing in benefits, paid holidays, and national insurance, the fact remains that large numbers of industrial workers have lost joy in their work. In a world which is so insecure it is not surprising that anxiety arising from feelings of instability should plunge into psychoneuroses those workpeople who are so predisposed.

Neurotic illness constitutes a group of disabilities covering over 90 per cent of psychiatric cases and is found in 30 per cent of the sick population of Great Britain. Such illness causes at least a third part of all absence from work due to illness and accounts for an average loss of work equal to three days a year per man and six days a year per woman. Factors tending to produce neurosis are long hours of work, inadequate diet, poor placement in which the job requires more skill than the operative possesses, together with extra-factory stresses related to the worker's social and domestic life. Neurosis is as common among skilled workers as among those in less skilled jobs. One characteristic of neurotic disability, considered as an occupational handicap, is its enduring quality.

Psychiatric handicaps unlike physical disabilities are not limited; they are so often personality handicaps, and as such they

fluctuate and may greatly interfere with retraining for any job at all. Where the neurotically ill are concerned, a precise diagnosis is less important than assessment of the personality and the morbid anomalies which the worker exhibits. Judgement of the latter is not beyond the competence of the industrial medical officer. It is he who holds the key position in giving psychiatric advice. He sees the workers, he has their confidence, and he knows his industry. By his contacts with workers exhibiting maladjustment he can discover where are the trouble spots in his industry.

Human Relationships in Industry

In the first half of the twentieth century industrial medicine has made an immense contribution to human health and happiness by its attitude to the group aspect of working life. Labour is more than a mass of individuals collected at random; economists and others have failed to grasp and interpret the importance of the group as an influence on the individual. It is mainly to the medical profession that we owe the concept of labour as a social group whose well-being is as important as that of any other group.

The study of the reactions of individuals and of groups of individuals to their work, their conditions of work, and their working environment, though by no means the prerogative of doctors, nevertheless comes within the scope of occupational health. For this reason the training of the industrial medical officer should be increasingly orientated in this direction because of the practical contribution already made by workers in this field to the contentment and efficiency of occupational groups. Since 1918, the Industrial Health Research Board and the National Institute for Industrial Psychology have carried out research on subjects such as the effects of light, temperature, and hours of work on the reactions of individuals; vocational guidance and selection; training methods; the significance of sickness-absence and labour wastage; accident prevention; and human relationships in industry.

Professor Elton Mayo has said that whereas material efficiency has been increasing for 200 years, the capacity for working together has, in the same period, continually diminished, and this

thesis may sum up the vital need for further studies in the field. The practical experience gained by Service psychiatrists and psychologists in the Second World War is of importance to occupational health. The use of psychological methods in the British Army developed in the following main fields: personnel selection; improvement of working methods and conditions; training methods; morale and incentives.

To be effective the industrial medical officer must know as much as possible about the jobs in his occupational group, whether it be factory, coal mine, shipyard, or office. This knowledge is best provided for him by the method of *job analysis*. This has now developed into a highly technical subject and is normally carried out by specially trained people. It consists in an intensive study of men at work and accurate recording of the observations made there, together with consideration of facts which are relative to the job and which may be obtained from workers, supervisors, and technical experts.

For medical purposes job analysis can conveniently be divided into two parts – the job description or specification, and an analysis of the demands which the job normally makes upon the worker, together with a note of environmental factors which may influence him. The written description should give a vivid picture of what the worker does, how he does it, why he does it, and the skill required in doing it. Job analysis is used in industry, however, for purposes other than health or safety; it can form the basis of the determination of wage-rate systems and may be used in production planning. It is of value to the doctor mainly as an aid to the placement of workers and when interviewing workers who blame their jobs for causing some health impairment; it is also useful when advice on a suitable change of occupation is required, or when processes and jobs are potentially hazardous or in cases in which excessive fatigue or other harmful results on health may be produced by inefficient plant design.

The state of the labour market has a bearing upon the selection of manual workers for jobs. When there is unemployment, industry has a large choice of workers, a fact which, before the Second World War, retarded the general development of scientific methods of selecting personnel. Today in Great Britain there is an

apparent lack of man-power, and the prospect of redeployment of workers from non-essential jobs, at which they are skilled, to others in which they may appear as raw material, makes the time opportune for the introduction of planned schemes of selection with the aid of job analysis. Results of this will be a reduction in labour wastage and absenteeism, combined with greater efficiency of the individual and increased output. The need for improved methods of placing men in industry has been accentuated by the number of disabled and handicapped persons for whom it is necessary to find employment. A useful step towards a solution has been taken by the Ministry of Labour, which has introduced a special form for use in placing disabled people in industry.

There is potential scope for the use of job analysis by hospitals and doctors concerned with rehabilitation, provided that local industries could supply simple job descriptions. At present, the majority of doctors advise on employment questions as a direct result of the patient's own description of the job, and the accuracy of the description is frequently open to doubt. Job descriptions are probably the best means by which any doctor can learn about occupation, and written descriptions mean more if accompanied by a photograph of the man on the job. It must be clearly understood that job descriptions can only be prepared effectively by persons with special technical qualifications and experience; this is no amateur task to be undertaken lightly by a doctor. Here immediate difficulties arise. Who is to do the descriptions? What will be the cost to the firm? These questions should be kept in mind when an approach for help in this matter is made to management, as it must be made, by industrial medical officers with foresight.

Assistance in describing certain of the physical and environmental demands of jobs can reasonably be given by doctors with industrial experience. Here, matters such as the amount of lifting, fingering, handling, or stooping have to be assessed, together with factors such as the need for good vision, colour vision, good hearing, or the ability to feel. Assessment of the psychological or mental demands of jobs is a matter of much greater difficulty. Factors such as wage incentives, monotony, the potential satisfactions to be found in the job, and the opportunity for responsibility

and promotion have all to be considered. The psychological characteristics of the individual can be assessed, in part at least, by psychologists and by doctors trained in psychiatric methods. These characteristics include intelligence, aptitudes, personality traits, and social adequacy, as well as educational levels and technical skill; the matching of these against the equivalent demands of the job, however, is a matter the significance of which can be decided only after prolonged observation and experience.

Training of new entrants to industry, particularly of youths under apprenticeship schemes, is generally accepted in the engineering trades. At *Government Training Centres* men and women receive simple vocational training designed to fit them for future careers, largely in the semi-skilled occupations. Schemes for the training of foremen and supervisors are sponsored by the Ministry of Labour. These schemes have three phases: methods of job instruction, instruction in human relations, and leadership and instruction in efficiency – that is, in the development of simple, time-saving, and fatigue-reducing methods of production. Education and experience are equally necessary in industrial management. In Great Britain over 400,000 persons are engaged in managerial work, and an annual intake of some 12,000 recruits is required. The syllabus of training, which is comprehensive in the technical, business, and commercial fields, also covers such subjects as industrial psychology, including the psychology of individual differences, measurement of intelligence, attainments and special aptitudes, personality and character, testing of temperament, interview techniques, vocational guidance and selection, psychology of training, of work, of incentives, and of work study, and social psychology, including the human factor in industrial relations.

Efficiency and high productivity depend upon industrial morale no less than on mechanical equipment. Factors influencing morale are, among others, the development of security, discipline and disciplinary action, leadership, individual behaviour, conditions at work, facilities for joint consultation and the development of joint responsibility, and specific incentives. The causes of good or bad morale are not easily determined, but the root of the problem seems to lie in an attitude to work which is described as

economic irresponsibility. This attitude may be derived from the economic and industrial structure of a community; it is certainly closely related to political and social factors outside industry.

Some of the symptoms of lowered morale are indifference to output and to the achievement and fate of the enterprise for which a man works, failure of the individual to understand the significance of his own work to the community, suspicion of the motives of the management, and the belief that the interests of labour and management are diametrically opposed to each other. These points have to be frankly discussed before any formal consultative machinery in industry can be made to work. Habits of mutual trust and consultation before decisions are made are fundamental for democracy in industry. The ultimate function of joint consultation is to get rid of the division of factories into bosses and the rest, which still colours the thoughts of most people. The wrong impression of management is traceable to the fact that some people when given responsibility do not like to consult the people under them.

Additional evidence of low morale is shown when workers demand security and control of industry but refuse to share the risks of industrial enterprise. They pursue their own sectional claims regardless of the effect of such behaviour upon the national economy. This is a menace to the national well-being and is incompatible with political responsibility and good citizenship. Yet a change of attitude on the part of labour cannot be expected unless there is a modification of those features in the industrial structure which are incompatible with a democratic way of life. The psychological effect on workpeople of having been consulted is more important as a rule than is the actual contribution of ideas made by them. Many employers are still insufficiently aware that the working group is a miniature society, and that, for its proper functioning, the art of social management is as important as is the technical management of machinery and processes.

The experiment carried out in 1938 at the Hawthorne Works of the Western Electric Company in the United States of America is perhaps the most significant contribution yet made to the problem of determining morale in industry. The immediate lesson appears to be that we have to recognize and respect those work-

relationships which develop within groups in most organizations, whether large or small. Groups develop an identity of their own which can enable them to take over some of the functions of supervision and to set behaviour standards which are at least as high as most employers would demand. On the other hand, they can develop, among employees, attitudes towards work which are based on misunderstandings due to lack of correct information. From this, awkward situations may arise in which groups faithfully believe something to be true which is, in fact, untrue. The *Hawthorne experiment* showed that improvement in conditions of work, discussed and explained to workers, gave rise to increased output. A more important point, however, was that the increased range of output continued when a return was made to the original imperfect conditions. These results were fully confirmed. The group had obviously developed some inherent force and had unconsciously, but yet effectively, organized itself for the specific task before it.

Probably the strongest motive for working is a man's desire to secure his own livelihood and that of his family. There is the lesser motive of a desire or urge to create. Also, there is the determination of a man to achieve distinction among his fellows. These motives are strengthened and reinforced by schemes and devices, at the place of employment, to which the term incentive is applied. The incentive has the power of awakening, maintaining, and strengthening the motive. Satisfying incentive systems will improve morale, and in these the basic is that of finance. There are other incentives, however, and some of these have been outlined by presupposing the existence of certain qualities, such as pride in rendering a service useful to the community, satisfaction in doing a job well, contentment through working in security with a competent and trusted chief, stimulation conditioned by opportunity for promotion, fulfilment arising from the performance of creative or constructive work, responsibility developing from opportunity to take the initiative, and a sense of participation arising from being consulted in matters of management. The application of these incentives in industry would do much to improve relationships and to encourage mutual trust between management and employees. Opportunities of personal

satisfaction for the individual would be regained, and harmony promoted more readily than through the defensive partisan approach of worker groups.

The National Insurance (Industrial Injuries) Act, 1946

The present national schemes for insurance against industrial injury began on 5 July 1948. Of all the post-war social schemes they were probably the most revolutionary. To understand the full implications of the new legislation it is necessary to appreciate some of the principles on which the *Workmen's Compensation Acts (1897–1945)* operated. The new Act has of course replaced the Workmen's Compensation Acts. Before 1897 an injured worker could recover damages only at common law, or, in certain cases, under the *Employers' Liability Act, 1880*. In either case, the worker was required to establish negligence to succeed in his claim. The Act of 1897, and subsequent amending Acts, established the principle, irrespective of negligence, of the employer's responsibility for the payment of compensation. But claims remained subject to individual litigation, and the case law built up made the Workmen's Compensation Acts so complex as to cause many anomalies and much bewilderment. The new scheme of industrial injuries insurance sets out to remove those anomalies.

Everyone employed under a contract of service or apprenticeship is insured under the *National Insurance (Industrial Injuries) Act, 1946*, and generally also under the *National Insurance Act*. The Industrial Injuries Acts insure people against incapacity, disablement, or death due to accident at work or to certain industrial diseases. Part of the weekly insurance stamp contribution is earmarked for industrial injuries. The amounts paid are:

	MEN		WOMEN	
	18 or over	*under 18*	*18 or over*	*under 18*
Paid by employee	5d.	3d.	3d.	2d.
Paid by employer	6d.	3d.	4d.	2d.
	11d.	6d.	7d.	4d.

These contributions, plus an Exchequer supplement, are paid into a special fund. There are no age limits for the Industrial Injuries Scheme and contributions are compulsory. Benefits do not depend on having paid a certain number of contributions and, save for the special hardship allowance, are not based on loss of earnings, as they partly were under the Workmen's Compensation Acts. They are the same for both men and women, but boys and girls get a lower rate. Benefits are of three kinds: injury benefit, payable for an initial period of incapacity after the accident or development of the disease; disablement benefit, where there is disablement, whether permanent or not; and death benefit.

Injury benefit is payable for incapacity during the first 26 weeks after an accident or development of an industrial disease and so covers in the vast majority of cases the whole period during which the insured man or woman is incapable of work through the injury. This benefit is 67s. 6d. a week with 25s. for a wife or other adult dependant, 11s. 6d. for the first child under school-leaving age, and 3s. 6d. for each younger child. It is paid in addition to family allowances. *Disablement benefit* is payable if the person suffers through the accident or disease any *loss of physical or mental faculty* including disfigurement. Disablement pensions range from 67s. 6d. a week for 100 per cent disablement to 13s. 6d. a week for 20 per cent disablement. For a life award of less than 20 per cent, down to 1 per cent, a gratuity varying from £225 to £22 10s. is paid. For the prescribed diseases, pneumoconiosis and byssinosis, neither injury benefit nor disablement gratuities are payable; where benefit is payable it takes the form of a pension.

The rate of benefit thus depends on the degree of disablement, and this is assessed by an *Industrial Injuries Medical Board* consisting of two or more doctors. The first assessment is normally for a limited period and further assessments may follow. On the matter of the assessment there is an appeal to a medical appeal tribunal. Each medical appeal tribunal consists of a legal chairman and two doctors of consultant rank. No allowance for dependants is payable with disablement benefit, except where an unemployability supplement is paid or the beneficiary is in hos-

pital for treatment for his injury. If there is treatment in hospital, the pension or gratuity is raised to the 100 per cent rate of pension. Disablement pension is not affected by any earnings of the claimant after the accident, even if they are as much as or more than before. A man getting basic disablement benefit can also get sickness or unemployment benefit. Of course, if no disablement remains when injury benefit ceases, which is the position in most cases, no disablement benefit is payable. On the other hand, if disablement grows worse a man can apply for a review of his case.

If a man has to take work different from that which he was doing before the accident he may get a *special hardship allowance* of up to 27s. 6d. a week unless he could do work of a standard equivalent to his old job. But the disablement pension and special hardship allowance together must not exceed 67s. 6d. What about the man who, because of his disability, will never be able to go back to any work? The Act provides for him a special allowance of 40s. a week with allowances for dependants, which can be paid in addition to the pension. This allowance is called an *unemployability supplement*, and a man entitled to it is one who is never likely to be able to earn more than £52 a year. His average earnings, therefore, could be 20s. a week and he could still get the 40s. allowance. If a man's disablement is assessed at 100 per cent and his injuries are such that he requires the attendance of some person to look after him, a *constant attendance allowance* of up to 30s. can be paid even if his own wife is attending him; in cases of exceptionally severe disablement, up to 60s. may be paid. A man with a 100 per cent pension who was entitled to unemployability supplement and constant attendance allowances would therefore be paid 137s. 6d. a week, or in cases of exceptionally severe disablement 176s. 6d. with perhaps further allowances of 36s. 6d. for a wife and child.

If a worker suffers an accident at his work, he, or someone for him, should tell his employer at once. In factories, mines, quarries, and at larger business premises an accident book is kept for this purpose. Claims are decided, not by the Minister of Pensions and National Insurance, but by authorities independent of the Minister. The claim is considered initially by an Insurance Officer, who decides whether or not benefit is payable. If the claimant is

dissatisfied, he can appeal to an independent local tribunal or, on the question whether he is suffering from a prescribed disease, to a medical board. In some cases, there may be a further appeal to the Commissioner appointed by the Crown, whose decision is final. As already stated, medical questions on industrial disablement benefit are decided by medical boards.

In case of death a weekly pension is paid to a widow who was living with her husband or being maintained by him. The amount is 55s. for the first 13 weeks after widowhood, followed by 45s. a week if the widow is over 50, or incapable of self-support, or if she has a child. In the latter case, the 45s. pension will continue after she ceases to have a qualifying child in her family, provided that she is over 40 years of age at that time. There is an addition of 11s. 6d. a week for the first child and 3s. 6d. a week for other children apart from family allowances. Where there is no child, a widow under 50 who is capable of self-support will get 20s. a week. These pensions are for life, unless the widow remarries, in which case she will get one year's pension as a gratuity. A pension of 45s. a week is also paid to the widower of a woman who dies from industrial injury, if he was being maintained by her and is not capable of supporting himself. In addition, the present scheme provides pensions, allowances, and gratuities for parents, certain relatives, and a woman having care of a child of the deceased, according to the extent of their dependence on the dead person. All these benefits are called *industrial death benefits*.

Prescribed Industrial Diseases

In Great Britain a worker who develops one of the 40 industrial diseases, as well as pneumoconiosis and byssinosis, prescribed by regulations is entitled to industrial injuries benefit, in the same way as a person who has met with an industrial accident, if he has been insurably employed in a prescribed occupation on or after 5 July 1948, and the disease is due to the nature of his employment. The list of prescribed diseases is extended from time to time as evidence accumulates to justify this. The question whether a person is suffering from a prescribed industrial disease is a medical one, and decisions given by medical boards, usually of

two doctors, are final, though boards may review their decisions if there is fresh evidence. As under the Workmen's Compensation Acts, special arrangements apply to the industrial lung diseases, pneumoconiosis and byssinosis; the medical questions are for decision by Pneumoconiosis Medical Boards consisting of specially qualified doctors. The Boards are stationed at several centres throughout the country.

Provision has been made by way of supplementary schemes for the payment out of the Industrial Injuries Fund of certain benefits and allowances for dependants, to persons disabled before 5 July 1948. The *Workmen's Compensation Supplementation Scheme, 1951*, provides for supplementary payments to workmen who are receiving low rates of compensation for accidents which occurred before 1924, and the *Pneumoconiosis and Byssinosis Benefit Scheme* provides benefits for workmen who are disabled by pneumoconiosis or byssinosis, but who are entitled neither to workmen's compensation nor industrial injury benefits. It also provides for the dependants of certain workmen who have died from either disease.

Prescribed Diseases Regulations, 1948

FIRST SCHEDULE, PART 1

Description of disease or injury	*Nature of occupation*
Poisoning by:	*Any occupation involving:*
1. Lead	The use or handling of, or exposure to the fumes, dust, or vapour of, lead or a compound of lead, or a substance containing lead.
2. Manganese	The use or handling of, or exposure to the fumes, dust, or vapour of, manganese or a compound of manganese, or a substance containing manganese.
3. Phosphorus	The use or handling of, or exposure to the fumes, dust, or vapour of, phosphorus or a

Description of disease or injury	*Nature of occupation*
	compound of phosphorus, or a substance containing phosphorus.
Poisoning by:	*Any occupation involving:*
4. Arsenic	The use or handling of, or exposure to the fumes, dust, or vapour of, arsenic or a compound of arsenic, or a substance containing arsenic.
5. Mercury	The use or handling of, or exposure to the fumes, dust, or vapour of, mercury or a compound of mercury, or a substance containing mercury.
6. Carbon bisulphide	The use or handling of, or exposure to the fumes or vapour of, carbon bisulphide, or a substance containing carbon bisulphide.
7. Benzene or a homologue	The use or handling of, or exposure to the fumes of, or vapour containing, benzene or any of its homologues.
8. A nitro- or amino-derivative of benzene or of a homologue of benzene	The use or handling of, or exposure to the fumes of, or vapour containing, a nitro- or amino-derivative of benzene or of a homologue of benzene.
9. Dinitrophenol or a homologue	The use or handling of, or exposure to the fumes of, or vapour containing, dinitrophenol or any of its homologues.
10. Tetrachlorethane	The use or handling of, or exposure to the fumes of, or vapour containing, tetrachlorethane.
11. Tri-cresyl phosphate	The use or handling of, or exposure to the fumes of, or vapour containing, tri-cresyl phosphate.

Description of disease or injury	*Nature of occupation*
Poisoning by:	*Any occupation involving:*
12. Tri-phenyl phosphate	The use or handling of, or exposure to the fumes of, or vapour containing, tri-phenyl phosphate.
13. Diethylene dioxide (dioxan)	The use or handling of, or exposure to the fumes of, or vapour containing, diethylene dioxide (dioxan).
14. Methyl bromide	The use or handling of, or exposure to the fumes of, or vapour containing, methyl bromide.
15. Chlorinated naphthalene (excluding the condition known as chlor-acne)	The use or handling of, or exposure to the fumes of, or dust or vapour containing, chlorinated naphthalene.
16. Nickel carbonyl	Exposure to nickel carbonyl gas.
17. Nitrous fumes	The use or handling of nitric acid or exposure to nitrous fumes.
18. *Gonioma kamassi* (African boxwood)	The manipulation of *Gonioma kamassi* or any process in or incidental to the manufacture of articles therefrom.
19. Anthrax	The handling of wool, hair, bristles, hides, or skins or other animal products or residues, or contact with animals infected with anthrax.
20. Glanders	Contact with equine animals or their carcases.
21. Infection by leptospira icterohaemorrhagiae	Work in rat-infested places.
22. Ankylostomiasis	Work in or about a mine.
23. (*a*) Ulceration of the corneal surface of the eye, (*b*) Localized new growth of the skin, papillomatous or keratotic,	

Description of disease or injury	*Nature of occupation*
	Any occupation involving:
(*c*) Epitheliomatous cancer or ulceration of the skin, due in any case to tar, pitch, bitumen, mineral oil (including paraffin), soot, or any compound, product, or residue of any of these substances	The use or handling of, or exposure to tar, pitch, bitumen, mineral oil (including paraffin), soot, or any compound, product, or residue of any of these substances.
24. (*a*) Chrome ulceration	The use or handling of chromic acid, chromate or bichromate of ammonium, potassium, sodium, or zinc, or any preparation or solution containing any of these substances.
(*b*) Inflammation or ulceration of the skin or of the mucous membrane of the upper respiratory passages or mouth produced by dust, liquid, or vapour (including the condition known as chlor-acne but excluding chrome ulceration)	Exposure to dust, liquid, or vapour.
25. Inflammation, ulceration, or malignant disease of the skin or subcutaneous tissues or of the bones, or leukaemia, or anaemia of the aplastic type, due to x-rays, ionizing particles, radium, or other radioactive substance; or inflammation of the skin due to other forms of radiant energy	Exposure to x-rays, ionizing particles, radium, or other radioactive substance or other forms of radiant energy.
26. Cataract produced by exposure to the glare of, or rays	Frequent or prolonged exposure to the glare of, or rays from,

Description of disease or injury	*Nature of occupation*
	Any occupation involving:
from, molten glass or molten or red-hot metal	molten glass or molten or red-hot metal.
27. Compressed-air illness	Subjection to compressed air.
28. Telegraphist's cramp	The use of Morse-key telegraphic instruments for prolonged periods.
29. Writer's cramp	Hand-writing for prolonged periods.
30. Twister's cramp	The twisting of cotton or woollen (including worsted) yarn.
31. Subcutaneous cellulitis of the hand (Beat hand)	Manual labour causing severe or prolonged friction or pressure on the hand.
32. Subcutaneous cellulitis or acute bursitis arising at or about the knee (Beat knee)	Manual labour causing severe or prolonged friction or pressure at or about the knee.
33. Subcutaneous cellulitis or acute bursitis arising at or about the elbow (Beat elbow)	Manual labour causing severe or prolonged friction or pressure at or about the elbow.
34. Inflammation of the synovial lining of the wrist joint and tendon sheaths	Manual labour, or frequent or repeated movements of the hand or wrist.
35. Miner's nystagmus	Work in or about a mine.
36. Poisoning by beryllium	Any occupation involving the use, or handling of, or exposure to the fumes, dust or vapour of, beryllium or a compound of beryllium, or a substance containing beryllium.
37. (*a*) Carcinoma of the mucous membrane of the nose or associated air sinuses, (*b*) Primary carcinoma of a bronchus or of a lung	Any occupation in a factory where nickel is produced by decomposition of a gaseous nickel compound which involves work in or about a building or buildings where that process or any other industrial process ancillary or incidental thereto is carried on.

Description of disease or injury	*Nature of occupation*
38. Tuberculosis	Any occupation involving close and frequent contact with a source or sources of tuberculous infection by reason of employment – (*a*) in the medical treatment or nursing of a person or persons suffering from tuberculosis, or in a service ancillary to such treatment or nursing; (*b*) in attendance upon a person or persons suffering from tuberculosis, or in a service ancillary to such treatment or nursing; (*c*) as a research worker engaged in research in connexion with tuberculosis; (*d*) as a laboratory worker, pathologist, or post-mortem worker, where the occupation involves working with material which is a source of tuberculous infection, or in an occupation ancillary to such employment.
39. Primary neoplasm of the epithelial lining of the urinary bladder (Papilloma of the bladder)	(*a*) work in a building in which any of the following substances is produced for commercial purposes: (i) *alpha* - naphthylamine, *beta* - naphthylamine, or benzidine, or any of their salts; (ii) auramine or magenta; (*b*) the use or handling of any of the substances men-

Description of disease or injury	*Nature of occupation*
	tioned in sub-paragraph (i) of paragraph (*a*), or work in a process in which any such substance is used or handled or is liberated; (*c*) the maintenance or cleaning of any plant or machinery used in any such process as is mentioned in paragraph (*b*), or the cleaning of clothing used in any such building as is mentioned in paragraph (*a*) if such clothing is cleaned within the works of which the building forms a part or in a laundry maintained and used solely in connexion with such works.
40. Poisoning by cadmium	Exposure to cadmium fumes.

FIRST SCHEDULE, PART 2

Pneumoconiosis. Fibrosis of the lungs due to silica dust, asbestos dust, or other dust, and including the condition of the lungs known as dust-reticulation.

1. Any occupation involving:

(*a*) the mining, quarrying, or working of silica rock or the working of dried quartzose sand or any dry deposit or dry residue of silica or any dry admixture containing such materials (including any occupation in which any of the aforesaid operations are carried out incidentally to the mining or quarrying of other minerals or the manufacture of articles containing crushed or ground silica rock);

(*b*) the handling of any of the materials specified in the foregoing sub-paragraph in or incidental to any of the operations mentioned therein, or substantial exposure to the dust arising from such operations.

2. Any occupation involving the breaking, crushing, or grinding of flint or the working or handling of broken, crushed, or ground flint

or materials containing such flint, or substantial exposure to the dust arising from any of such operations.

3. Any occupation involving sand blasting by means of compressed air with the use of quartzose sand or crushed silica rock or flint, or substantial exposure to the dust arising from such sand blasting.
4. Any occupation involving:

   (*a*) the freeing of steel castings from adherent siliceous substance;

   (*b*) the blasting of metal castings to free them from adherent siliceous substance by means of any abrasive, by a blast of compressed air, by steam, or by a wheel;

   (*c*) the moulding of iron castings with the use of siliceous materials as a facing powder or parting powder;

   (*d*) substantial exposure to the dust arising from any of the foregoing operations.
5. Any occupation in or incidental to the manufacture of china or earthenware (including sanitary earthenware, electrical earthenware, and earthenware tiles), and any occupation involving substantial exposure to the dust arising therefrom.
6. Any occupation involving the grinding of mineral graphite, or substantial exposure to the dust arising from such grinding.
7. Any occupation involving the dressing of granite or any igneous rock by masons or the crushing of such materials, or substantial exposure to the dust arising from such operations.
8. Any occupations involving the use, or preparation for use, of a grindstone, or substantial exposure to the dust arising therefrom.
9. Any occupation involving:

   (*a*) the working or handling of asbestos or any admixture of asbestos;

   (*b*) the manufacture or repair of asbestos textiles or other articles containing or composed of asbestos;

   (*c*) the cleaning of any machinery or plant used in any of the foregoing operations and of any chambers, fixtures, and appliances for the collection of asbestos dust;

   (*d*) substantial exposure to the dust arising from any of the foregoing operations.
10. Any occupation involving:

    (*a*) work underground in any coal, tin, slate, or haematite iron-ore mine;

    (*b*) the working or handling above ground at any coal or tin mine of any minerals extracted therefrom, or any operation incidental thereto;

(*c*) the trimming of coal in any ship, barge, or lighter, or in any dock or harbour or at any wharf or quay;

(*d*) the sawing, splitting, or dressing of slate, or any operation incidental thereto.

Byssinosis. Employment for a period or periods amounting in the aggregate to not less than ten years, in any occupation in any room where any process up to and including the carding process is performed in factories in which the spinning or manipulation of raw or waste cotton is carried on.

CHAPTER FOUR

ACCIDENTS AT WORK

The Causes of Accidents – Accident Proneness – Prevention of Accidents – Accidents at Docks, Wharves, and Railway Sidings – Hazards of Work in Sewers – Treatment of Industrial Injuries – Dust Explosions in Factories – Accidents in Mines and Quarries

IT is common knowledge that a high proportion of all accidents occurs in the home and on the roads. In Great Britain factories, of course, contribute their share of accidents. But as well as accidents in factories we must consider also accidents in mines, quarries, and sewers, at docks, wharves, and railway sidings; and also firedamp and dust explosions in coal mines, dust explosions in factories, accidents in agricultural work, and electrical injuries. The annual report of the Chief Inspector of Factories for 1955 records the lowest accident-rate yet achieved in factories, although the number of accidents, 188,403, was 1·2 per cent greater than in 1954, the factory population being 2·5 per cent higher and the number of factories with power 0·4 per cent greater than in 1954. Since 1953 the accident-rate for women has increased, and the Chief Inspector attributes this largely to the general rise in the number of women employed, of whom many are new to industry. He stresses the responsibility of management for special care to protect those entering industry for the first time and completely unfamiliar with their environment, as well as the importance of proper induction, training, and supervision for young persons.

THE CAUSES OF ACCIDENTS

Accidents in factories can be classified as due to the handling of goods, power-driven machinery and lifting machinery, to persons falling and blows by a falling body, the use of hand tools, stepping on or striking against objects, to burns from molten metal, and to transport. Of the fatal accidents, nearly a third are due to

falls, sometimes from a height, but often while walking on the level. Many of these mishaps are attributable to lack of suitable appliances, such as ladders, or the wearing of unsuitable shoes, especially by women. Slippery floors and imperfectly guarded gaps in the floor or walls lead to many accidental falls. The mishandling of goods is responsible for over a quarter of the non-fatal accidents and is often associated with faulty supervision, or with the employment of persons who lack the necessary experience, strength, or agility to do the work safely. Accidents continue to occur frequently through falls through roofs made of fragile material such as asbestos sheeting or glass. Such accidents are often due to carelessness, and entail a high mortality, about 20 per cent. Many accidents on building sites and in engineering construction can be traced to faulty methods of stacking and storing materials; here, as in factories, there is need for greater study of good housekeeping.

In Great Britain accidents to the eyes occurring in industry number on an average 7000 a year. For certain processes, especially in the chemical industry, the provision and use of goggles is compulsory, and in a number of other processes they have been introduced by voluntary action on the part of the employers or adopted by the workers themselves. The processes mainly responsible for these accidents are the grinding, glazing, and polishing of metal articles; the pouring, stirring, and carrying of molten metal (Plate 6); riveting, dressing castings, chipping metal articles, smithing, metal working by machinery, and stone dressing. The provision of an adjustable transparent screen between the worker and his work is an alternative which has been found successful. Where possible, arrangements should be made for the repair of goggles at the actual place of work. Unhappily the goggles-service man is an economic possibility only in firms employing large numbers of men.

As a serious industrial problem *photophthalmia* is of importance only in electric arc welding and in gas welding by means of oxy-hydrogen, oxy-coal gas, and oxy-acetylene torches. For this reason it is often referred to as *arc eye*, *arc flash*, or *electric-arc welders' eye-flash*. Although experienced welders are rarely affected, their mates and even passers-by are often less fortunate.

It may also arise from gazing at the intense glare of molten steel, from the examination of furnaces and hearths in power plants, from the failure to screen the arc lamps in cinema studios, and from exposure to the arcs formed by electrical apparatus equipped with ineffective switch gear. Beyond the momentary glare and dazzle there is no immediate effect of exposure to intense ultra-violet radiation. A few hours later, however, the victim may observe coloured rings around lights; these arise from oedema of the corneal epithelium. This symptom may be followed by pain and a feeling as of grit in the eyes, excessive secretion of tears, and intolerance to light. Fear of blindness may lead to emotional upset. The eyes become suffused and there may be marked spasm of the eyelids. The injury responds to simple treatment such as eye drops of liquid paraffin. In all occupations presenting this hazard goggles, eye-shields, visors, or screens of Crookes' glass must be provided and properly used (Plate 1).

We can accept without further discussion that the occurrence of accidents is influenced by such factors as hours of work, speed of output, temperature, and lighting, the inexperience or youth of the workers, and the injudicious use of alcohol. However well machinery may be guarded by law, a reduction of not more than 10 per cent in the accident-rate can be looked for by the provision of safeguards alone. Psychological factors in the causation of accidents have been systematically studied in England since 1917 under the direction of what was at first the Industrial Fatigue Research Board and is now called the Industrial Health Research Board. The important fact that a minority of workers is responsible for more than their proper share of accidents, if not actually a discovery of the Board's investigators, was first established by them upon a statistical basis.

Fatigue of the worker in industry involves psychological factors such as boredom. Lack of incentives to work, and the presence of opportunities for divided attention, may reduce the workers' efficiency and must therefore be recognized and studied. Many changes have taken place in industry since the replacement of man-power by machines. With the reduction of working hours from ten to eight, excessive muscular fatigue is less important, and we are now concerned more with the workers' mental and

nervous reactions to their environment. Accidents which were formerly charged against fatigue are now attributed to the personal equation. A predisposition to accidents may result from lack of training or from an abnormal attitude towards the work. Noise should receive attention, because it leads to nervousness and a feeling of weariness similar to that produced by muscular fatigue.

The benefit of rest periods lies chiefly in the reanimation of interest. In addition, the change in posture or occupation involved permits the blood to circulate more freely to those parts of the body which were not receiving their proper supply in a cramped position at the machine or bench. The mental and nervous reactions of workers to their environment as causes of fatigue and so of accidents will increase in importance with the passing of time. No matter how well working conditions may be controlled, such factors as the home life and psychological make-up of the individual worker will remain and become increasingly more important.

Accident Proneness

An examination of almost any large collection of accident statistics shows that accidents are very unevenly distributed among those exposed to risk. With equal exposure to risk, roughly three-quarters of recorded accidents happen to one-quarter of the people exposed. This phenomenon is known as *accident proneness*, a term coined by Farmer in 1932 and now in world-wide use. The probable number of persons who are accident-prone ranges from 10 to 25 per cent of the total. Unfortunately, few firms subject their new employees to psychological tests before assigning them to one occupation or another. Even in the absence of such tests it would be possible to sift them roughly by putting them for a time on occupations free from major risks, and only after they have shown themselves to be reasonably free from accident proneness to transfer them to more dangerous occupations. Accident proneness is greatly influenced by the mental attitude of the subjects. The accident-prone are apt to be insubordinate, temperamentally excitable, and to show a tendency to get flustered in an

emergency. These and other defects of personality indicate a lack of aptitude on the part of the subjects for their occupation.

It would clearly be desirable to devise means by which accident proneness can be detected before exposure to industrial hazards. It was found that accident proneness was generally associated with some impairment of neuro-muscular control, and that certain psychological tests could be used to measure such control, as it affected attention, muscular precision, and co-ordination of movement. A bad record in these tests did not necessarily imply that the subject would show a high susceptibility to accident, but some very significant associations were established. Thus, in a group of 1800 engineering apprentices subjected to these tests, whose records at work were afterwards examined, it was found that, if they were arranged in order of proficiency at these tests, the quarter fraction at the bottom of the list subsequently sustained accidents at work at a rate which was two and a half times as high as that in the remaining three-quarters of the group. It is clear that if this quarter had been put into some less dangerous occupation, the incidence of accidents in the whole group would have been greatly diminished. There are no large-scale records available to show the effect of the early detection of accident-prone workers and their transfer to safer jobs, but this procedure has been put into operation in a few firms.

Turning to the individual, the problem is more complex. Divided attention may be due to emotional influences such as injustices imagined or real, quarrels with colleagues or at home, and love affairs. Simple tests for accident proneness can be illustrated by quoting the case of motor drivers. The National Institute of Industrial Psychology devised a series of tests for the purpose of directly measuring certain qualities regarded as essential for safe and efficient motor driving. Reaction time, resistance to distraction, vigilance, judgement of speed, judgement of spatial relationships, confidence, road behaviour, vision – these and other qualities are given separate marking, the final judgement resting upon the sum of the reactions. This method is satisfactory up to a point, for those who come out badly in the tests are shown by their records to be liable to an excess of accidents.

But the disadvantage of such tests is that they cannot repro-

duce the conditions of long-distance driving – they are too transverse; what is required is something more longitudinal. As in the case of intelligence tests, they are adequate only for the short period of the test, not for longer periods when the attention is less held. In the competent motor driver we find a fluid state of mind which diminishes fatigue. He is watching all the time, not only his own road but side roads, and his judgement is continually operating. This includes judgement of the speed of vehicles coming towards him both in front and behind. His psychological make-up is important; the too-careful driver may be almost as bad as the reckless driver. When motor drivers are interviewed, the accident-prone driver can often be picked out. A single bad habit in driving, susceptibility to a small dose of alcohol, wilful road-hogging, loss of sleep, family worries, lack of correct instruction, are all known causes. It is claimed that psychophysiological selection before training has reduced the proportion of unsuitable trainees for the buses of Paris from 20 per cent to 3·4 per cent.

Accidents of every sort, in greater number than the public would ever guess, are of psychological origin. Ranging from insignificant mishaps like stumbling, bumping oneself, or burning the fingers to motor accidents and catastrophes in the mountains, instances may be found of psychological causation. In motor driving some men have an impulse to charge at an oncoming vehicle, and have to give up driving. Some drivers when interviewed will admit the existence of an impulse which makes it impossible for them to drive behind another car when they cannot see the road ahead of it. They are thus impelled to draw out and try to pass. An obsessional impulse to speed is found in some people.

Such impulses and inhibitions can now be detected by the method of clinical interview. Here a knowledge of disease and symptomatology is, of course, necessary in order that the clues given by the patient may be followed up. An interviewer seeking for nervous symptoms or temperamental qualities must know what to look for and how to look for it. The use of the method shows that about 20 per cent of the working population suffer from nervous symptoms which interfere with happiness and

efficiency. Preventable accidents thus constitute a problem of human behaviour, and in feeling our way to an understanding of the behaviour problems concerned we are reaching out to new means for the prevention of accidents.

In 1954 an advance in the understanding of individual accident susceptibility was made by Whitfield. He analysed information on underground *compensation accidents* in relation to individual working records at a Nottinghamshire colliery employing approximately 2000 men. By classifying the accidents and the shifts worked according to occupation and place of work it was possible to calculate the accident risk per shift for moderately homogeneous working groups. From these figures an expected total of accidents could be computed for each miner and compared with the actual number experienced. The results indicated clearly that there were individual differences in accident susceptibility among coal-miners which persisted for several years, but that these were not readily attributable to the age of the worker or to inherited tendencies. A detailed investigation was therefore made of three small samples of the miners. One was composed of men who had experienced many more accidents than were expected, the accident-prone group. Each individual was then matched, by age and normal mining occupation, with two others who had experienced, respectively, about as many accidents as were expected and fewer accidents than were expected.

Thirty-two of these triads were examined by interview and a series of tests; the panel which tested each triad did not know which member belonged to which accident category. Among the tests used were measurements of visual and auditory acuities, performance tests of perceptual, cognitive, and memory functions, and tests of motor control and co-ordination. It was found that the younger accident-prone men were markedly deficient in the perceptual-cognitive tests, while the older accident-prone men were markedly deficient in motor control and co-ordination. Whitfield suggests that the younger accident-prone man is unable to appreciate the demands of a hazardous situation or to decide what response should be made, although capable of making the response, whereas the older accident-prone individual fails to produce an adequate response, even though the hazard has been

perceived and an appropriate course of action initiated. If this is so, it is easy to understand why those naturally deficient in motor ability when young should become accident-prone as their motor ability slowly deteriorates with age. It is not so easy to see why young accident-prone miners should apparently cease to be so. Some, presumably, die young; the rest perhaps leave the industry for less hazardous occupations, or, while remaining in coal-mining, adjust themselves to a reduced level of activity.

Prevention of Accidents

To protect a machine by mechanical guards is a simple and obvious procedure, but teaching good housekeeping and safe conduct in work-places is a slow process of education both of workers and supervisors. Safety regulations are restricted to general indications and cannot provide for particular cases. There must be organized a system of prevention considered from the human aspect with provision for individual selection of workers on entering industry and periodical medical supervision. This is especially important in those trades which expose the workers to special causes of fatigue and to sustained attention. All this is very much the concern of the doctor, and it is admitted that medical officers in industry have not paid sufficient attention to the prevention of accidents.

The Regulations under the Factories Acts have reduced the workers' liability to accidents, and, in order still further to forward the work, the Factory Department of the Ministry of Labour maintains in Horseferry Road, London, the *Safety, Health, and Welfare Museum*, where various types of safety devices can be seen and studied. During 1950 there were slightly more than 12,000 visitors to the Museum; most of them were in organized parties numbering from six to fifty persons. More than half of these parties came from factory groups, trade schools, technical schools, medical schools, and universities. In 1951 one group comprised all seventy members of the safety committee of a large factory in Birmingham, while another consisted of fifty-five doctors taking a post-graduate course in public health. Each year some hundreds of people come by appointment to discuss

problems regarding existing machinery and future extensions to plant and factories.

Transmission machinery is responsible for many accidents of the most serious kind. They are caused by the workers coming into contact with the revolving shafting or other moving parts of machinery while engaged in oiling bearings, adjusting belts, cleaning shafting, executing repairs, or performing other work in the vicinity. The danger of revolving shafting cannot be over-estimated.

If loose clothing, such as an apron, a coat end, a wide or ragged sleeve, or a rag in the hand of a cleaner, comes into contact with the shaft, it may wrap round the shaft in a moment and, before the wearer is aware of what is happening, he may be caught, whirled round, and killed or terribly injured. Such accidents can and do occur even with smooth shafting; the contrary view often expressed is contradicted by experience. Moving belts are also responsible for many accidents, in some of which the worker is caught and carried round the shaft. Every kind of transmission machinery is a potential cause of accidents, and it is required by the Factory Acts that all such machinery be securely fenced or be in such a position or of such construction as to be equally safe. Whenever possible, machines should be driven directly by a motor or engine so as to eliminate transmission machinery.

Machine tools are responsible for numbers of accidents. Many of them are due to the absence of efficient guards upon dangerous parts such as toothed gears, chain gears, belts, and pulleys. Many machine-tool makers have designed their machines so as to avoid the exposure of dangerous parts – for example, by the complete enclosure of gear wheels. The dies or tools of power presses have long been recognized as dangerous. Accidents are generally severe and usually result in permanent mutilation of the hand. Much thought and ingenuity have been devoted to the problem of protecting the operator. This has been made more difficult by the great variety of materials dealt with and the many different operations performed.

Guards for power presses are of many kinds, including fixed, automatic, and interlocked types. The guard most suitable for the job in hand must be carefully considered in each case, and it

is important that it should be impossible for the machine operator to displace or circumvent the guard and that it should cause the minimum possible interference with output. When machine operatives are paid on a piece-work basis, they may increase their earnings by reducing the time taken in removing completed work from their machine and in setting up new work. The time taken in moving the machine guard after each operation may tempt the operative to dispense with the guard in an effort to increase production.

Unfortunately, certain workers are loath to use safety devices because they regard them as unworthy of an experienced worker and only fit for the young and inept. They feel that there is something unprofessional about them, in the same way that a barber will not demean himself by using a safety razor. Coercion is of little avail against such a feeling, for in trying to combat it we shall be up against that pride of achievement which is a man's chief incentive to action. If, however, we can show that true courage manifests itself more in putting up with the discomfort of a guard and so preserving a life that is useful to others than in running unnecessary risks, we are more likely to get the workers to use the guard provided than if coercion alone is attempted. In all industries, most production machines introduce risk in varying degree, and much effort has been devoted to guarding the dangerous parts to reduce to a minimum the chance of an accident occurring.

In the much wider field of accidents which are not caused by machinery, physical safeguards play an important part, but constant care and good housekeeping are essential. This applies particularly in the case where workers fall or where objects fall on to them. In factories, special pathways indicated by two parallel white lines painted on the floor should be kept clear of any object whatsoever at all times.

In certain occupations protective clothing is of great value. Where its use is not obligatory it is often sold at reduced prices to the worker. Strong stiff hats, rubber aprons, coats, overalls, shin guards, rubber boots, gloves, and goggles are standard articles used widely in handling chemical substances and in the mining industry. Where possible, equipment such as safety shoes should be handled in a special department. Reinforcement of the

toecap by a steel arch is an excellent protective device for use in heavy industry (Plate 2). Drilling machines are the main cause of hair-entanglement accidents, of which 95 per cent happen to women and girls and 5 per cent to men and boys. Women workers should protect themselves against such scalping accidents by wearing turbans which completely enclose their hair. The slightest projection, even a small grub screw, on the drill renders such an accident more likely, but it is important to teach the worker that a revolving spindle which is perfectly smooth may be equally dangerous in its effects. In the manufacture and handling of glass special machinery and protective clothing are necessary.

By means of propaganda and education the Safety Movement has done good work to reduce accidents. It has spread to all civilized countries, and is probably the most powerful influence at work today in accident prevention. It aims at protection through the education of the industrial worker by means of posters and lectures, advice to the employer as to safety methods, and the formation of Works Safety Committees. By means of posters placed in prominent positions in factories, the attention of the workers is called to the grave results of accidents which may be due to their inattention, carelessness, or failure to carry out instructions. The posters are changed from time to time so that they may attract fresh attention and keep alive the workers' interest in accident prevention. Such posters should not dwell unduly on the gruesome side of accidents lest they induce a state of morbid fear in the minds of those who see them. Fear is undoubtedly a strong deterrent, but it should not be used too much lest it produce a state of nervousness which may itself cause accidents.

One of the best methods of lessening accidents is the promotion of Works Safety Committees on which the workers, the management, and the works medical officer are all represented. These committees inquire into the causes and circumstances attending accidents, and by their intimate knowledge of the working conditions in the factory they are able to make wise recommendations to prevent a repetition of such accidents. They are also well fitted to consider the advisability of adopting any suggestions sent in by the workers as to means of preventing accidents.

The Royal Society for the Prevention of Accidents (RoSPA) and the Industrial Welfare Society have established many Area Committees to make inquiries and recommendations in such matters as prevention of sepsis; steps to promote the provision of a local Fracture Clinic; accidents to juveniles; effects of fumes from welding; breathing apparatus and gassing; eye injuries and use of goggles; traffic accidents; and electrical safeguards. These Committees and their parent Societies also carry on a campaign to reduce accidents by educational methods, exhibition of posters, conferences, lectures, competitions, and award of prizes.

Doctors, engineers, and physicists must constantly collaborate in efforts to improve schemes of safety and to devise new methods to protect the worker against injury. Certain automatic engineering devices which guarantee safety are now commonplace. Thus metal stamping machines are guarded in such a way that the electric circuit which operates the mechanism cannot be closed unless the worker is outside the guard and the guard locked. A safety unit has been developed which consists of a radioactive wristband worn by a machine operator and a detection instrument on the machine. If the operator's hand enters a dangerous area, the radiation emitted from the wristband operates a control which stops the machine.

Accidents at Docks, Wharves, and Railway Sidings

A dock is an enclosed water area with facilities for the handling of ships, their passengers, and their cargoes. There are two sorts, wet and dry. Wet docks are of two kinds, enclosed or open. In the enclosed kind, the water is impounded, and such a dock is commonly found where there is a great rise and fall. At Sunderland and Bristol, the main dock systems are enclosed; at Southampton and Glasgow they are open. Dry docks or graving docks are used by vessels undergoing survey or requiring underwater repairs.

Wharves are to be found along the containing walls of wet docks. They are also variously known as quays, jetties, berths, or staiths. Wharves may be open or they may be equipped with

transit sheds, gear stores, offices, and other ancillary buildings. They may have quay cranes or they may not. There will be bollards or mooring rings, probably railway lines, possibly one or more capstans, very often hydrants, and sometimes pipe connexions and valve pits for handling bulk oil. They may have any one of many different types of special equipment for special trades. Alongside the wharf there will be a sufficient minimum depth of water to accommodate the type and size of vessel for which the wharf is designed. There will normally be some form of lighting to enable work to proceed in hours of darkness. Railway sidings at docks include exchange sidings, sorting sidings, running lines, quayside tracks, standage sidings, light roads, and gravitation sidings.

The danger that men will fall into docks and get drowned or injured is always present and is greater of course in foggy weather. Cranes sometimes topple over with great peril to the drivers and other persons. Railway wagons being shunted or moved may get out of control. Motorists, pedestrians, and cyclists are sometimes impatient and take foolish risks when crossing dock railway lines. Slinging of cargo is sometimes badly done, so that heavy objects fall from the slings, trays, or nets when cranes are working. Cargo-handling gear is sometimes faulty or ill-chosen, so that the load collapses in transit between ship and shore. Dock workers handling cargo are liable to injury moving in or out, or up and down, between the shore and the bottom of ships' holds. Gantry girders have been known to collapse, with great peril to crane drivers and to persons working below them. A winch-handle slipping out of control with a load on the fall puts the operator and others in grave danger. A slipping foot on the brake pedal of a lifting appliance similarly creates a dangerous situation for all concerned. A crane driver may be killed when he suddenly brakes to avoid dropping a heavy load on persons working below him. The ballasting of the backstays of derricks has been known to fail, involving the collapse of the whole appliance. Drivers can be trapped and drowned by the heavy weight of their own protective equipment if the full safety drill is allowed to fail in any particular. It is perhaps especially true of dock undertakings that inevitably a large amount of general maintenance is always going on at the

same time as the everyday work; ships must be turned round; cargo must be moved between ship and shore; any lack of liaison at all necessary levels, between the traffic manager on the one hand and the engineer on the other, is therefore extremely likely to produce dangerous conditions.

To get as near to perfect safety as is humanly possible the following conditions should be present. First, all the requirements for safe working should be borne in mind from the beginning and incorporated in the design and layout of the facilities themselves. Secondly, similar requirements should mark the design, strength, and fitness of all mechanical appliances, tools, and gear. Thirdly, the facilities and the equipment must be continuously and properly maintained and regularly tested. Fourthly, entirely safe methods of working must be devised for all operations and must be efficiently taught to all operators concerned or likely to be concerned. And fifthly, the operators must use the safe methods and no others. Over the past 150 years scores of millions of pounds have been spent in building docks, wharves, and railway sidings in the United Kingdom; in many cases, the broad lines of the original layout remain to this day. Unhappily with increased mechanization and modern changes in speed, method, and purpose, the existing conditions are not always the best for safe working. Nevertheless, great opportunities to increase safety in layout do occur; extensions, improvements, and modernization schemes are always going on; and, in particular, since 1945 the dock authorities in Great Britain have not been slow to grasp the special opportunity presented to them as the result of enemy action during the war, when damage in the docks, spread over 33 ports, amounted to £28,000,000.

Transcending protective restrictions, regulations, and prohibitions is the mental attitude of the management, the supervisors, and the workpeople to the whole problem of accidents. They must ever have in mind that men matter more than machines and more than money. The management must plan wisely, skilfully, and safely; and they must appoint supervisors who will never, in their enthusiasm for the work, lose sight of the human beings upon whom the work depends. And finally, the workpeople themselves, knowing they are respected by those who direct the

work, will respect themselves and one another, and, in consequence, there will grow among them a spirit of thoughtfulness instead of carelessness, cheerful alacrity instead of sluggish compliance, and keen-eyed watchfulness instead of the dangerous inattentiveness which produces more accidents than any other single cause.

Hazards of Work in Sewers

Following the disastrous epidemics of cholera which raged in London killing 20,000 people between 1831 and 1849 Sir Edwin Chadwick insisted on the construction of a main drainage scheme for London. Sir Joseph Bazalgette in 1855 started work on a system which took 20 years to complete. The centenary of this work was celebrated in London in 1955. Since 1855 the population had increased from 2¼ to 4½ millions of people. In 1955, during the working hours of any one day in London, drainage facilities were used by some 7 million people, and the daily consumption of water per head had gone up in 100 years from 20 to 50 gallons.

The length of main sewers has increased from 163 to 403 miles. In the London main drainage area there are in addition some 3000 miles of sewers maintained by local authorities. The sewage now treated is 103,336,000,000 gallons per annum, and 1,777,150 tons of sludge are sent to sea, to Black Deep 60 miles out from the mouth of the Thames. A similar state of affairs applies in other large towns; for example, Glasgow maintains 800 miles of public and common sewers which flow into the large intercepting sewers. Men working in sewers are liable to accidents causing physical injury, to drowning, and to gassing accidents, including the explosion of mixtures of air with methane or petrol. The hazard of Weil's disease as it applies to sewer workers is discussed on p. 224.

The sewers in which men work are usually 4 ft. 6 in. or more in vertical diameter. In order to be self-flushing they are egg-shaped. Usually the sewer flusher wades through water about ten inches deep and walks on a varying amount of solid silt. He wears

leather-soled, hob-nailed, rubber thigh-boots and gloves of cotton cloth. Accidents leading to physica injury occur from falls from ladders and foot-irons. Drowning is not unknown. Floods may result from storms, and the main drainage systems in towns usually include storm-relief sewers. No man may enter a sewer alone, and there must always be a man stationed on top of the manhole at the place where work is being carried out. Of course nobody works in a sewer while it is raining. The top man looks out for rain and when it begins to fall he signals to the men below by slamming down the manhole cover three times or by rattling a crowbar in the grating. Sounds of this sort travel a long way in the sewer. As soon as the men hear the signal they know they have but a few minutes to get out.

Safety chains or iron bars are provided at the side entrance immediately below where the men are to work, and these must be fixed across the sewer by the first man who enters it. In fast-flowing sewage or when the work is otherwise dangerous the men should be roped together by life-lines. Arrangements must be made for the top-men to be told of any storms, high tides, or sudden large discharges from public baths, cooling tanks, ponds, or water mains, so that the men in the sewers can be called up before the flood wave reaches them. Care should be taken that the men have a way out downstream of their working place. In the event of a man being caught by a flood, provided the guard-bars or chains are in position, he can let himself go with the flood without much risk.

To work in a sewer is to enter a lonely world of inky darkness and deathly silence. The atmosphere feels damp and has a faint sickly musty odour sometimes replaced by a more penetrating smell such as that of tar, paraffin, petrol, benzene, or ammonia. Sewage is a mixture of liquids and solids of domestic and industrial origin which varies in composition from sewer to sewer and from hour to hour. Most of us think of sewage as being composed of kitchen water, bath water, and human excreta, but to the sewermen working in industrial towns trade wastes are the important constituents. Engineering works contribute oils and grease as well as pickling acid, cyanides, and suds, while garages are a common source of paraffin, petrol, and diesel oil. From

chemical works weak acid, weak alkali, spent carbide, resins, and other materials enter the sewers. Ammoniacal liquors containing phenol and tar compounds gain access from steel works and gas generators. The paper industry and wool-scouring factories produce alkalis and soaps, and many trades provide grease and other animal products. Although the discharge into sewers of most of these materials is prohibited by law, enforcement is difficult, and so they appear from time to time.

Inflammable and explosive gases and vapours occasionally found in sewers include coal-gas, methane, acetylene, petrol, and benzene. No man may take matches or a cigarette lighter into a sewer and no naked flame or fire is allowed within ten feet of any sewer entrance. To maintain ventilation during the course of work, the covers of the manholes upstream and downstream of the manhole from which work is in progress should be kept open. Under the general law special precautions must be taken to prevent the admission of petrol into sewers. Garages must have drains provided with petrol traps, but often they neglect to empty these. Such accidents as that resulting from a street collision during the transport of petrol in bulk may lead to an explosion. Arrangements should always be made to warn men in the sewers of such an accident. The sewer worker carries a flame safety lamp and he knows that the flame will cease to burn if he enters an area where there is insufficient oxygen to support life. He must be taught that even a small oxygen deficiency impairs co-ordination and leads to danger from failure to control the limbs accurately. He also uses the flame safety lamp to detect methane just as the coal miner does.

In sewers with a low velocity flow the atmosphere may become contaminated with hydrogen sulphide from the decomposition of deposits of sewer solids. Dangerous concentrations of this gas may occur when acid wastes from manufacturing processes are discharged into sewage containing sulphides. For detecting hydrogen sulphide the sewer worker is provided with lead acetate paper which he moistens and attaches to his flame safety lamp. Dangerous concentrations of hydrogen sulphide will blacken the paper. Hydrogen cyanide is a hazard in sewers, because cyanides may be present in gas-works effluents and in trade wastes dis-

charged from electroplating shops. Sewermen are instructed that should any unusual smell be met with in a sewer, especially a smell of almonds, all men shall leave the sewer at once and report to the superintendent. Unfortunately the almond odour of hydrogen cyanide can never be detected by numbers of people, including heavy smokers. In consequence its presence in sewers is a greater hazard than that of hydrogen sulphide. Other poisonous gases entering sewers from time to time are carbon dioxide, carbon monoxide from leaking gas mains, ammonia from refrigerator plants, chlorine, phosgene, nitrous fumes, sulphur dioxide, and carbon disulphide. Trouble can occur when removing sludge in which trichlorethylene or benzene has been entrapped.

Sewermen are generally of small build, cheery disposition, and almost fearless. They display a remarkable team spirit and have in general the qualities found in miners. What attracts them to the job is the certainty of continued employment and the higher rate of pay for skilled work. Usually they are recruited from the general labour force engaged in road repairs. They are observed at work for several years and when a vacancy arises in the sewage squads a man who has proved his worth is then selected. Among Glasgow sewermen all are married and most have at least three children, about 50 per cent of them living in overcrowded houses without a bath. Periodic medical examination is essential; the local authority should launder the working clothing and provide at the depots wash-basins, showers, baths, hot water, soap, and towels for all the men. Mechanical transport provided with proper facilities for washing in hot water should be a standard part of equipment. Rodent control should be improved in sewers and they should be better ventilated and better lighted. Electric handlamps should be issued in addition to flame safety lamps. The best electric lamp has two bulbs side by side, a white one for continuous use and a red one which lights up automatically in the presence of dangerous concentrations of toxic, asphyxiating, or explosive gases. Sewermen should not be condemned to unemployment at 55 after 20 years of faithful service in this arduous task. Local authorities should find them alternative work and devise superannuation schemes to provide for their future.

Treatment of Industrial Injuries

The objects of treatment in a case of industrial injury should be to secure the repair of the damaged part as quickly and completely as possible, and to do what may be necessary to replace the workman in remunerative employment, at his former job if possible, or in some other occupation, for which he may need training and assistance in placement.

The study of methods of first-aid and of the emergency surgery of injuries was greatly advanced during the Second World War, when much experience was gained of the efficacy of various methods of dealing on the spot with casualties from air raids and other forms of enemy action. In industry there may be special hazards when accidents occur from handling corrosive or toxic substances, or from electricity, radiant energy, or extremes of temperature or pressure. The St John Ambulance Association has recently published a text-book of industrial first-aid, in which special attention is given to these risks. *Section 45 of the Factories Act, 1937*, provides that in every factory –

> there shall be provided and maintained so as to be readily accessible a first-aid box or cupboard of the prescribed standard . . . under the charge of a responsible person who shall, in the case of a factory where more than fifty persons are employed, be trained in first-aid treatment.

There is no statutory definition of responsible person or of what is meant by 'trained in first-aid treatment'. In practice, the requirements of the Act are held to have been fulfilled if one or more of the workers in the factory holds a valid certificate in first-aid issued either by the British Red Cross Society or the St John Ambulance Association.

Emergency surgical treatment includes arrest of haemorrhage, blood transfusion, cleansing and suture of wounds, radiography of suspected fractures, and their reduction and fixation when present. Valuable time may be saved, and shock averted, by arranging for injured workers to be taken in a heated ambulance to a hospital at which the ambulance is driven directly into a heated resuscitation room equipped with all necessary apparatus

for giving such emergency treatment at any hour of the day or night.

The measures include the further treatment of wounds or fractures, and operations for repair of damaged nerves, correction of deformities, or elimination of infection. A particular industry may require the full-time services of a surgeon with special skill and experience, for example to suture the tendons at the wrist in the case of men who are injured while lifting and carrying sheets of plate glass.

Physiotherapy includes heat, massage, electrical treatment, and remedial exercises. In carrying out such treatment it is essential to secure the active co-operation of the patient. Physiotherapy commonly fails because it is carried out so infrequently. Thus a patient in order to reach hospital may have to travel fifteen minutes by train, and then half an hour by bus. He is therefore obliged to endure one and a half hour's cold in order to enjoy a quarter of an hour's hot air. At the Albert Dock Hospital in London, patients attend the gymnasium all day and every day until they recover full working capacity.

The word *rehabilitation* is used in various senses (see p. 56). It is commonly meant to imply the refitting of the patient, after his injuries are healed, for remunerative employment and his placement in a suitable job. This may entail the provision of an appliance such as an artificial limb, or a course of training in a new occupation. Difficulties often arise over resumption of work owing to the lack of proper co-operation between those responsible for treatment and failure to consider the industrial environment to which the patient should return. Either he is discharged from hospital before he is fit to undertake the strain of work, or he continues attendance for long after he has reached a stage at which work would be the best form of treatment. If industrial injuries are to be treated effectively, it is necessary for the surgeon to make himself familiar with the physical requirements of the industries in which his patients work. He must maintain as close a liaison as possible with the management of the local industries and with the officers of the local Employment Exchange who are concerned with vocational training, placement, and supervision. In these matters the surgeon will, of course, make full use of the

services of the almoner and other social welfare workers, since the proper care of a case of industrial injury calls for teamwork between those concerned with the medical, surgical, financial, social, and even the legal consequences of the accident.

Research into methods of treatment applicable to industry has come from the Unit of the Medical Research Council at the Birmingham Accident Hospital. It has included the design of dressing-stations and the control of wound infection, studies on added infection in industrial wounds, the incidence of sepsis in industry, and the bacterial flora of wounds and septic lesions of the hands. The treatment of burns has been especially studied at the Burns Research Unit of the Medical Research Council at the same hospital. Here, work of much significance has been carried out; death-rates have been reduced by special procedures in plastic surgery, and by the use of an air-conditioned dressing-station which has greatly decreased the incidence of septic infection.

Dust Explosions in Factories

Besides coal dust, many other combustible dusts may produce explosions if thrown into suspension in air and ignited. Such explosions may take place in a wide variety of establishments, including grain elevators, wood-working plants, cereal mills, flour mills, sugar refineries, fertilizer plants, malt houses, cotton mills, and plants producing starch products.

Explosions of combustible dusts may take place in plants where cork dust, pulverized coal, metal dust, sulphur dust, bark dust, coffee, cocoa and spice dusts, paper dust, gramophone-record dust, pitch and resin dust, rubber dust, and soap powder are present. As a result of the increased use of metal powders in powder metallurgy and in military pyrotechnics, there has been an increase in metal-dust explosions. Such metal powders include magnesium, aluminium, cadmium, zinc, copper, iron, manganese, titanium, ferromanganese, antimony, zirconium, and even tin and lead. Similarly, the number of explosions has increased in the rapidly expanding moulded plastics industry, in which many combustible synthetic resins, moulding compositions, and fillers are used in powder form.

A dust explosion may be defined as the rapid combustion of a cloud or suspension of dust in air, during which heat is generated at a much higher rate than it is dissipated to the surroundings. This phenomenon, which is similar to that of a gas explosion, is characterized by the sudden development of pressure, which frequently causes the destruction of both the plant and the equipment containing the dust. The conditions necessary for the explosion are a sufficiently dense cloud and an ignition source intense enough to raise the temperature of part of the dust mixture to the ignition point.

The following have been established as some of the definite causes of industrial dust explosions: electricity, such as sparks from motors, fuses, switches, short-circuits, static electrical discharges, and the breaking of incandescent lights; frictional sparks from foreign materials going through grinding mills and into fans, from sledge hammers, from workmen's shoes, and from grinding wheels and buffers; hot particles, such as glowing material fed to mills or dust collectors and sparks from boiler fires and locomotives; heated surfaces, such as overhead bearings and other moving parts of machines, dust which has settled on hot light-bulbs, dust on steam coils and on hot pipes in driers, friction in grain elevators; open flames and lights, such as lanterns, candles, gas-lights, torches, matches, and smoking, and miscellaneous small-scale fires, including spontaneous ignition of waste and other materials, breaks in fuel lines, and boiler backfires; small explosions of inflammable vapours and of dust blown into furnace and incinerator flues; and the disturbance of burning dust by the use of a water-hose.

Accidents in Mines and Quarries

In spite of efforts to reduce accidents in mines, mining remains amongst the most dangerous of our major industries. This is due to the natural conditions under which mining work is carried out, which have hitherto made it impossible to reduce the accident-rate to the average of that in other industries. In quarrying also, for similar reasons, the accident-rate remains high. The *Mines and Quarries Act, 1954*, which replaced the *Coal Mines Act, 1911*,

tightened up the existing safety law and brought it into line with modern mining and quarrying practice.

Great Britain is essentially a coal-mining country; the mining of tin and lead have been for years constantly diminishing. The only remaining branch of metalliferous mining is that for iron ore, and much of this is done by opencast methods. Quarrying is carried out to obtain alabaster, chalk, chert, flint, granite, graphite, gravel, gypsum, kaolin, limestone, sand, sandstone, and slate. The hazards met by the quarry worker include falls of ground, detachment of rock, blasting accidents, falls from ladders or ledges, eye injuries from the dressing of stone, and accidents connected with tramways, the breaking of ropes or chains, and the mismanagement of machinery.

The *Statistical Digest of the Ministry of Fuel and Power* sets forth annually the story of an immense amount of human suffering and loss of output due to accidents in mines. There is, however, a bright side to the story, since during the last quarter of a century or so there has been a marked reduction in the number of men killed and seriously injured in our coal mines. This gratifying improvement has been specially noticeable during recent years. The total number of accidents in 1955 was the lowest recorded since 1949. The number of men killed during 1953 and 1954 was the lowest on record. The year 1955 unfortunately showed an increase, 421 against 392 and 371 respectively for all mines worked under the *Coal Mines Act*. From 1945 to 1947 there was a tendency for the number of comparatively minor accidents, causing disablement for more than three days, to decrease. This tendency was, however, reversed during the period 1948 to 1952, but it again decreased from 1953 to 1955, although the figure did not reach that recorded in 1945. Intensive campaigns are carried out amongst the miners with the object of persuading them to adopt precautions against sepsis following minor injuries. Continuous efforts are made to improve the lighting in the pits and, of course, miners wear protective clothing.

It is incumbent upon all engaged in mining – workmen, management, trade-union leaders, inspectors, and research workers alike – to do everything possible to attain that further improvement necessary to reduce the accident-rate, and to keep

British mining in the proud place it has so long held, as the safest in the world. In this connexion it is often pointed out by H.M. Inspectors of Mines and others concerned with the safety of men working underground that many accidents would be avoided by the exercise of proper care on the part of the injured persons. This does not imply that all the avoidable accidents are due to gross errors or major and wilful contravention of the regulations. In many cases they arise from a commendable desire to obtain production even at the expense of safety, such as when a man continues to get or to load coal at the face instead of attending to support of the roof, with the result that he is injured by a comparatively small fall which might easily have been prevented. Not infrequently, however, accidents occur as the result of lack of forethought, carelessness, or even recklessness on the part of miners. If every worker underground took proper precautions at all times, the accident-rate in mines could be reduced to one-half of the present rate. This would mean not only a reduction in human suffering, but also an increase in output well worth the effort demanded.

Falls of ground are responsible for about 50 per cent of the fatal accidents in coal mines, and over 40 per cent of the serious and non-fatal accidents. A large proportion of accidents from falls occurs under freshly exposed roof at or near the working face, where the excavation is newly made and the supports are usually of a temporary character. About 80 per cent of the accidents due to falls occur at or within 10 yards of the working face. Many of these accidents occur at the vicinity of the road-head. It is customary in this area to enlarge the roadways by ripping down the roof at what is called the ripping lip. As this lip is being constantly carried forward and exists in ground which is badly broken and still in the process of settling down, support of the roof calls for special care. This is shown by the fact that about 50 per cent of the accidents from falls in or near the face occur in this area. The precautions against accidents due to falls at or near the working-face include rigid compliance with the regulations and careful inspection and testing of the roof at frequent intervals. The early and systematic setting of all supports – sprags, props, chocks, and packs – in accordance with the support rules

is of the utmost importance; neglect and delay allow the roof to subside and become broken, making it difficult to support.

Many of the supports at or near the coal face are of a temporary character and have to be withdrawn as the face advances. This applies especially to sprags, props, and chocks, which are withdrawn, not only to conserve supplies of materials, but also to ensure effective roof control in the vicinity of the coal face and roadheads. The operation of withdrawing supports especially in packholes and wastes is skilled work, and calls for training, care, and experience, as it is attended by a certain amount of danger. It should be undertaken only by qualified workers fully conversant with the correct methods of withdrawal practices. The *Coal Mines* (*Support of Roof and Sides*) *Regulations, 1947*, require that all workmen should be adequately trained in the work they have to perform.

Something like 15 per cent of the accidents from falls occur in mine roadways. About half of these happen to men employed in repairing and enlarging the roadways, and the remainder to men employed on other duties or travelling on the roads. Accidents from falls on roadways are reduced by special care in the formation and support of the roadway at the time it is made and by efficient roof control at the face. The importance of the support and control of ripping lips and roadheads cannot be overemphasized. Approximately one-third of the fatal accidents caused by falls of ground occur in this vicinity. Good roof control reduces the amount of fracturing of the roof strata and better and safer roads result. Although subsidence of the roof behind the face cannot be prevented, it can be controlled and the strata caused to subside without being unduly broken. In many cases the use of yielding supports, such as arches on stilts or sliding-girder arches, affords the safest means of supporting roads in the moving ground near the face. These yielding supports are removed and rigid supports inserted when the ground has finally settled.

After falls of ground, the next most serious source of accidents in mines arises from injuries to men employed in haulage operations. Haulage is responsible for about 25 per cent of the total fatal and serious non-fatal accidents and for more than 20 per

cent of all accidents. Although prior to 1938 the accident-rate due to haulage had remained almost stationary for over fifty years, since that date a substantial reduction in the serious-accident rate has been recorded.

Haulage accidents arise from many causes; about 70 per cent occur to men during the actual performance of haulage operations, about 10 per cent to men travelling on haulage roads. The greatest number is due to men being crushed or run over by moving traffic, and runaways, and this is responsible for about 80 per cent of the deaths and upwards of 50 per cent of all accidents due to haulage. Restricted space and inadequate lighting have been the principal contributory factors. Comparatively few serious accidents are caused by breakages of ropes or draw gear. This is due to the careful choice and thorough examination of ropes or draw gear in practice, and to detailed investigations made into all cases when failure of these appliances occurs. Such investigations often suggest remedial measures.

The reduction in serious accidents due to haulage has been effected largely by detailed study of the problem by the various Mining Institutes in conjunction with the Safety in Mines Research Board, the National Coal Board, and by the circulation of illustrated pamphlets in which the causes of haulage accidents have been analysed, examples of safety devices and precautions described, and recommendations made for increased safety in haulage operations. The prevention of such accidents calls for attention to many details. Special safety devices such as runaway switches or tub arresters should always be employed where there is danger of runaways. Such switches should be designed either to fall to safety or to be automatic. Backstays or drags should be attached to all sets ascending inclines, and should be of such a design that they cannot fall off or be overridden. Skotches and wheel lockers which do not fall out accidentally and which can be inserted without danger of the operator's fingers being trapped should be used. Coupling blocks should be hung between tubs or cars during coupling and uncoupling to avoid danger of injury to the operator's head if placed inadvertently between the tubs or cars, a practice which should always be avoided. Good track is essential to eliminate derailments, which are a common source of

accidents. The provision of refuge holes, adequate means of lighting, efficient signalling equipment, the prohibition or limitation of travelling on haulage roads when vehicles are in motion, and the avoidance of interference with signals and other apparatus by unauthorized persons are other essential requirements for the safe and efficient running of haulages.

Owing to the more extensive use of trunk conveyors and locomotive haulage, there is reason to expect a considerable reduction in haulage accidents in newly opened and in reconstructed mines. The use of locomotives necessitates flat gradients, larger road clearances, and improved tracks, more in line with surface railway practice, all of which tend to increase safety. The use of large mine-cars and the reduction of the number of men employed in haulage operations, together with the better lighting of haulage roadways, all will have a beneficial effect. These, in conjunction with adequate training of all haulage operatives in conformity with the regulations, should make underground haulage as safe and efficient as surface haulage. Not only should the accident-rate be reduced by these means, but conditions of work will be vastly improved and manual labour considerably reduced.

The *First-aid Regulations, 1930*, call for adequate provision to be made at all mines for rendering first-aid in case of accident and for subsequent treatment of all injured persons. At every mine employing more than a hundred persons on a single shift, a suitable first-aid room, adequately equipped, must be provided at the surface, and must be in constant charge of a competent person. It is also required that a sufficient number of the persons employed below ground shall hold certificates of proficiency in first-aid. Thus one person in every fifty in each district in charge of a deputy, and one in every thirty employed elsewhere, shall be so qualified. Dressings and antiseptics must either be carried by each workman, or in first-aid boxes, one of which must be carried by each first-aid man. In the latter case the first-aid boxes must be taken to the surface at the end of the shift and replenished as necessary. First-aid stations equipped with stretchers, splints, bandages, tourniquets, and burn dressings must be provided at convenient places underground throughout the mine. Ambulance

service must be available, and the manager must inspect the accommodation, equipment, and materials at least once in every six months and see that defects, if any, are remedied.

The *Mines Rescue Regulations, 1928*, require that adequate provision for rescue shall be made at all mines employing more than ten men underground. For this purpose, properly equipped Central Rescue Stations must be provided. Each such station must be under the control of a fully trained and competent superintendent, must be within 15 miles of the mines served, and each mine must be in constant telephonic communication with the station. Central Rescue Stations are organized in two distinct ways: first, those which maintain a permanent rescue corps, similar to the staff of a fire station, ready for immediate action as required, and secondly, those which train rescue brigades from the collieries served by the station. In the latter case, when a disaster occurs the necessary rescue apparatus is brought from the Rescue Station and rescue teams are organized from the trained men at the colliery, generally under the supervision of the Rescue Station Superintendent, who also makes arrangements for the testing and servicing of the rescue apparatus.

In both cases a specified number of trained men, depending upon the number employed below ground, must be employed at each colliery. Self-contained breathing apparatus, which enables the wearer to enter and work in irrespirable atmospheres, is employed. It is of two types – the liquid-air type and the oxygen type. In both cases purifiers, which allow exhaled air to be rebreathed, are employed to conserve the liquid air or oxygen, and each type suffices for use for a period of two hours. The Mines Rescue Regulations specify in detail the methods to be adopted in the selection and training of rescue workers, the equipment to be provided and the general organization of rescue operations. Rescue Brigades deal with fires in mines and perform valuable rescue work after explosions and other accidents, often in circumstances of extreme danger. No praise can be too high for these men who volunteer for the work and are always ready to face peril in valiant attempts to save life and property.

Among the total coal-mining population of Great Britain it is estimated that there are about 250,000 accidents per year, each

causing absence of more than three days, a startling and appalling figure. In 1951, among a population of 106,000 coal miners, injuries to the limbs accounted for 61 per cent of such accidents, the upper limb being involved in 36 per cent and the lower limb in 25 per cent of cases. The problem of permanent paraplegia from spinal injury presents great difficulties. In time of peace 90 per cent of all such injuries occurring in Great Britain are due to accidents to miners while working underground. Indeed, the incidence of paraplegia in coal miners has been calculated at one case per 10,000 miners per annum. Since these men cannot be nursed at home, special institutions have been built for their treatment and rehabilitation.

The *National Coal Board Medical Service* is a comprehensive industrial health service for all employees of the National Coal Board. The Medical Service is an independent department of the Board represented at headquarters and in the divisions and areas. It is headed by a medical officer in each case. In the larger areas the area medical officer has an assistant. State-registered nurses are employed at the larger collieries. By 1956 there were 70 medical officers in the Service, including three engaged on medical research, and about 300 nurses. The Medical Service carries out the following functions:

(i) *Pre-employment Examination of Workers:* Virtually all new entrants to the mining industry are examined by the Medical Service. In some coalfields the examination includes a radiograph of the chest. By the end of 1958 a regulation will be in force making radiographic examination of the chest universal.

(ii) *Advising Management on the Hygiene of the Working Environment:* It is particularly difficult to secure good hygienic conditions in coal mines. Unlike the factory worker, whose working conditions remain much the same from day to day, the miner is continually battling with changing conditions. Lighting, dust, and ventilation, as well as heat and humidity in deep mines, all present challenging environmental problems.

(iii) *Advising Management on the Medical Aspects of Safety:* Careful pre-employment examination can make an obvious contribution to safety. Further, the study of mining jobs by doctors in the industry, who have a special knowledge of the

psychological and physiological problems involved, should make for safer working.

(iv) *Organization of First Aid:* Owing to the dangerous nature of mining operations the accident rate in coal mines is higher than in any other major industry. A highly efficient first-aid organization is thus essential if loss of life and disability are to be kept as low as possible. There are some 20,000 volunteer first-aid men in British coal mines and a considerable effort is required to assure that their knowledge of first aid is kept up to date.

(v) *Treatment of Injury and Illness:* The first treatment of men sustaining an injury or becoming ill at work is a responsibility of the Medical Service. At the larger pits treatment is given at medical centres staffed by State-registered nurses. The smaller pits have good first-aid rooms staffed by medical-room attendants.

(vi) *Resettlement of Miners after Injury or Illness:* The miners' rehabilitation units provide an excellent rehabilitation service, the importance of which needs no emphasis. The service is extended by a follow-up when the convalescent miner returns to the pit. In addition to cases due to injury, men suffering from pneumoconiosis require regular supervision.

(vii) *Research:* Fundamental research on miners' diseases is, generally speaking, carried out by units of the Medical Research Council. Applied research, on the other hand, is the responsibility of the Medical Service. This research is carried out either directly by the research staff of the Medical Service or indirectly by selected departments in certain medical schools which receive grants of assistance from the Board.

The provision of pithead baths at all collieries where the life of the colliery justifies it is nearing completion. The National Coal Board, through the agency of the Coal Industry Housing Association set up in 1952, have carried out a housing programme in those areas where more houses were urgently wanted than the local authorities could provide, and some 20,000 houses have been built in well laid-out estates. The *Coal Industry Social Welfare Organization* has continued the activities of the Miners' Welfare Commission in the social welfare field, and in addition to the provision of further community centres and playing fields they have stressed the encouragement of sporting and cultural activ-

ities. The Organization has undertaken a survey into the social needs of 550 paraplegic mineworkers, and holiday schemes and other amenities have been provided for these men. It is intended to extend the existing welfare service to other mineworkers who are seriously disabled whether through accident or disease. Meanwhile British miners are proud and appreciative of their rehabilitation service which is the best of its kind in the world.

CHAPTER FIVE

POISONING BY METALS AND THEIR COMPOUNDS

Lead – Mercury – Arsenic – Manganese – Nickel – Chromium – Beryllium – Cadmium – Vanadium

NATURALLY some metals have been in use much longer than others; lead, mercury, and arsenic have been recognized as poisonous since ancient times. Of all the new metals discovered later on, only some came into use at once. Of the metals which have been introduced into industry in our own time a good deal is known about the effects of chromium, manganese, nickel, radium, and thorium, and the toxicity of many others is at present under study. So far no toxic effect has been discovered in workers handling caesium, cerium, columbium (niobium), gallium, germanium, indium, molybdenum, rhenium, titanium, wolfram (tungsten), and zirconium. On the contrary, toxic effects are already known in the case of beryllium, cadmium, osmium, platinum, selenium, tellurium, thallium, uranium, vanadium, and a large number of radioactive compounds.

A metal may enter the tissues without acting as a poison. Thus silver produces no toxic symptoms although it can bring about lifelong disfigurement in the form of generalized argyria. A metal or a compound of a metal may have a different effect according to whether it exists in organic or inorganic form, whether its physical properties are those of a solid, a liquid, or a gas, whether the valency of the metal radical is high or low, or whether it falls upon the skin or enters the body through the respiratory or alimentary tract.

POISONING BY LEAD

Lead poisoning may occur in industry in two forms: (*a*) from exposure to the inorganic compounds of lead, and (*b*) from handling organic compounds, especially tetra-ethyl lead. The clinical

picture is different in the two forms. Poisoning by the inorganic compounds causes colic, wrist-drop, stippling of the red cells, and anaemia. In poisoning by tetra-ethyl lead the picture is that of insomnia, mental confusion, delirium, and mania. Only poisoning by the inorganic compounds of lead will be considered here.

Man has been using lead for at least 6000 years. It is a soft, bluish-grey metal, heavy, malleable, and ductile. Bright metallic lead is not dangerous to handle, but exposed to the air it readily becomes coated with a film of oxide. This creates dust which is inhaled by men who shovel scrap lead. Further, lead readily volatilizes at higher temperatures, and lead smelting and all lead-melting and lead-burning jobs are hazardous. Other industrial processes which may give rise to lead poisoning are vitreous enamelling on glass or metal, glazing of pottery, manufacture of lead compounds such as litharge, red lead, white lead, and lead colours, manufacture of lead accumulators, shipbuilding and ship-breaking, painting, plumbing, soldering, and the manufacture of rubber.

Hippocrates (370 B.C.) described a severe attack of colic in a man who extracted metals, and was probably the first of the ancients to recognize lead as the cause of the symptoms. Although lead be absorbed into the tissues it does not always cause poisoning. Since it is a common constituent of soil, and hence of animal and vegetable foods, its presence in traces in the human body is not unusual. A distinction must be made between lead absorption and lead poisoning. A diagnosis of lead poisoning is not justified unless there are signs and symptoms of actual illness. Lead taken by mouth is mostly excreted in the faeces: that fraction which is absorbed is taken up by the liver and most of it is returned to the intestine in the bile. Lead absorbed through the respiratory tract on the other hand passes directly into the circulation, by-passing the liver, so that much smaller doses may produce symptoms. In industry lead poisoning is almost invariably the result of inhaling dust or fume containing lead. Inorganic lead compounds are not absorbed through the skin, but the opposite is the case with organic compounds such as tetra-ethyl lead. When lead is being absorbed rapidly into the body, it becomes widely distributed, and

the question of whether or not it will cause symptoms of illness depends on individual susceptibility and on the ratio between the rate of absorption and the rate of excretion. When absorption is slow and continuous over a long period of time, lead is deposited in the calcareous portion of the bones as the insoluble tertiary phosphate. There it causes no symptoms. The metabolism of lead runs parallel to that of calcium, so that conditions which favour the storage of calcium in the bones also favour the storage of lead. Conversely, stored lead is remobilized and returned to circulation by conditions, such as a depletion of the alkali reserve, which alter the reaction of the body fluids. It is for these reasons that single figures for lead excretion taken from spot samples of urine are difficult to interpret.

Individual opinion differs widely as to what is necessary for the *diagnosis* of lead poisoning. Constipation and slight stippling of red cells are insufficient; neither a blue line on the gums nor detection of lead in the urine can be taken as proof of poisoning, for the patient may be insusceptible. Where a worker is exposed to risk, a diagnosis of lead poisoning can be made before the occurrence of a toxic crisis. A falling haemoglobin percentage, with or without a rising punctate count, raises a suspicion that absorption is passing into poisoning. This suspicion becomes a certainty when these changes are marked or progressive. The diagnosis offers no difficulty in the presence of colic, palsy, anaemia, or encephalopathy.

Intestinal colic is the commonest manifestation of plumbism. It is ten times as common as lead palsy. An attack of colic is preceded by several days of constipation. The pain is situated around or below the umbilicus. The patient indicates where it is by spreading both hands widely over the abdomen. He becomes cold, pale, and drenched with perspiration, and may bend over or writhe in bed in intense pain. Examination reveals a scaphoid abdomen showing no rigidity. Vomiting may occur at the onset of the pain.

Encephalopathy is the most dramatic and the most dangerous manifestation of lead poisoning. It begins suddenly with an epileptiform convulsion. There may be also attacks of coma, delirium, convulsions, transient paresis, aphasia, and anaesthesia.

Chronic cases may show mental dullness, inability to concentrate, poor memory, headache, head retraction, trembling, deafness, transitory aphasia and hemianopia, and amaurosis without fundus changes.

The commonest form of *lead palsy* is wrist-drop, which begins on the right side in right-handed persons and later becomes bilateral. Paralysis does not appear to be related to the length of exposure. It may develop during the first month of work or only after many years' exposure. The palsy first appears in the long extensors of the middle and ring fingers. It spreads to the other fingers, and then to the long extensors of the wrist. The supinator longus escapes. The wrist-drop causes the flexors of the fingers to work at a mechanical disadvantage, which can be at once overcome by passive fixation of the wrist joint. Wasting flattens the posterior aspect of the forearm, throwing into relief the integrity of the supinator longus. Lead palsy rarely occurs in the lower limbs, but when it does it affects the extensors of the toes, giving rise to foot-drop. The paralysis is in the first instance a muscle disease. Fatigue plays an important part in determining the sites attacked. There is never any associated sensory loss nor does the paralysis show the symmetrical distribution of polyneuritis.

The *blue line on the gums* consists of fine granules of pigment, arranged in the form of a dark blue stippled line, within the tissue of the gum and about a millimetre from the gingival margin. It is more marked round teeth having infected gingival troughs and is more frequently seen on the mandibular gums than on the maxillary, and in the incisor region than in the molar. If there are no teeth it does not occur. It is a precipitate of lead sulphide caused by the action of hydrogen sulphide upon the lead salts in the circulation. The gas is formed by micro-organisms in infected gingival troughs. Despite the pigment lying within the tissues, careful cleansing of the mouth and teeth often causes it to disappear. It is significant of absorption and not of intoxication. Its intensity and size provide a rough guide to the duration and severity of exposure to lead.

Chronic lead poisoning may give rise to a mild *low colour-index anaemia*. It is therefore of value to estimate the haemoglobin figure in persons exposed. In differential diagnosis it is

important to remember that in any case of industrial plumbism it is rare to find less than 3,000,000 red cells per c.mm. Examination of the blood for *punctate basophilia* is of value in the prophylaxis of plumbism and, indeed, essential in the hygienic control of lead processes if the occurrence of manifest plumbism is to be avoided. The presence of basophilic stippling in the blood is not a specific sign of lead poisoning. It is seen in pernicious anaemia, leukaemia, the anaemias of carcinomatosis, in pneumonia in infants, and sometimes in normal persons. However, its occurrence in these conditions is rare and slight as compared with the frequency and intensity of its appearance during plumbism. The stippling is of cytoplasmic origin. The basophilic granules are independent of the nuclear substance and have their origin in the reticulum.

In Great Britain *preventive measures* against lead poisoning have been carried out with striking success. Originally based on the work of Sir Thomas Legge they can still be expressed in his aphorisms:

(i) Unless and until the employer has done everything – and everything means a good deal – the workman can do next to nothing to protect himself, although he is naturally willing enough to do his share.

(ii) If you can bring an influence to bear external to the workman (that is, one over which he can exercise no control), you will be successful.

(iii) Practically all industrial lead poisoning is due to the inhalation of dust and fume; and if you stop their inhalation you will stop the poisoning.

(iv) All workmen should be told something of the danger of the material with which they come into contact and not be left to find it out for themselves – sometimes at the cost of their lives.

(v) Examples of influences – useful up to a point, but not completely effective – which are not external, but depend on the will or whim of the worker to use them, are respirators, gloves, goggles, washing conveniences, and waterproof sandpaper.

As a result of Legge's work lead poisoning was made notifiable in 1899. Since then, despite a steady increase in the consumption of lead in the United Kingdom, a satisfactory decline has occurred

in the incidence of the disease. Thus 49 cases of lead poisoning were notified in 1956 as compared with 1058 in 1900.

Lead encephalopathy has disappeared from industry. It is unusual today to meet with a case of either severe colic or extensive palsy. Few cases of lead poisoning are seen and these are mild. Nearly all lead hazards can be controlled by engineering devices of which the most important single measure is exhaust ventilation applied through hoods at the source of origin of dust or fume. The hygiene of the workshops and cleanliness of the worker are both important. Benches, tools, floors, and walls must be spotless, often at the expense of the constant vigilance of several good foremen. No scrap lead or dry white lead should be handled unless it has been thoroughly soaked by the use of a hose. Mechanical means, such as cranes, rails, hoists, covered conveyors and hoppers, and automatic packing machinery, should be substituted for hand carriage (Plate 3). Between 1899 and 1939 the lead manufacturers in Great Britain spent over £200,000 on alterations and dust-removal plant in order to render the conditions of employment more healthy. Between 1930 and 1950 more than £250,000 was spent in changing over entirely from the old stack process to the quick process in the manufacture of white lead.

In the manufacture of electric accumulators, suppression of dust in the pasting shops is effectively carried out by the use of exhaust ventilation over the benches combined with repeated hosing with water of the benches and floors. The lead burner must be rigidly protected by a system of exhaust ventilation. This is arranged to operate behind a plate-glass screen so that the worker's nose and mouth are safely protected from lead fume; meanwhile he is able to see to do his work through the glass. Professor Ronald E. Lane, Nuffield Department of Occupational Health, Manchester University, has contributed work of great value in protecting workers in the electric-accumulator industry against lead poisoning.

In the printing trade the compositor keeps his type in cases. From time to time he removes the dust containing lead oxide from the surface of the type by the use of a vacuum cleaner. At one time it was customary for him to blow off this dust, and there was then a risk that he would inhale some of it. The hazard in the

case of linotype workers is slight only. Linotype metal is an alloy of 85 per cent lead, 12 per cent antimony, and 3 per cent tin. This is kept in the molten state in a container on the machine, but the temperature is low (350° C.) and the total quantity is only one gallon.

Since 1927 it has been illegal for a painter to rub down by dry methods any indoor structure previously treated with lead paint. Dust can be avoided by using a damp rubbing-down process for lead-painted surfaces. Waxed sand-paper, which the workman dips repeatedly in a bucket of water, has made this possible. This material is impervious to water, and it has been found possible to immerse it for six months without deterioration in the quality of the paper or loosening of the abrasive. The paper has a much longer working life than ordinary paper applied in the dry method, and this adds to its efficacy. Master painters agree that the damp process can be applied efficiently to all types of work, including the rubbing down of curved surfaces. Spray painting where the paint contains compounds of lead must be strictly forbidden.

In mixing paint a painter is rarely exposed to dry white lead since it comes to him already mixed in oil. Paint technologists have invented a non-setting red lead, which is issued to the painter of metals already mixed in oil. He is therefore protected from exposure to the dust of red lead, for he no longer mixes the materials himself. The setting properties of red-lead paints depend upon the proportions of true red lead and lead monoxide present, the latter being more active chemically towards linseed oil. It is now technically possible to produce commercial red lead having so little immediate action on the paint media that it can be ground with linseed oil into a paste which remains soft for many months. Originally, dry red lead as obtained by the painter contained such high proportions of the monoxide that it had to be mixed with linseed oil immediately before it was required for use, as otherwise it would set hard.

The use of litharge rubber – that is, rubber in which litharge has been incorporated in excess in a mother batch even to the extent of 90 per cent – has abolished lead poisoning in men who vulcanize rubber. The litharge rubber is manufactured in a central factory and is sent to scores of other factories where vulcanizers

throw it in solid pieces into batches of crude rubber. Prior to the invention of litharge rubber they used powdered litharge, some of which inevitably they inhaled.

At one time the occupation of pottery glazing exposed men to the hazard of lead poisoning. The potter glazes his ware with vitreous materials of various types. The glazes always contain silica, alumina, and at least one alkali or alkaline earth metal, and many contain up to 70 per cent of lead oxide. In pottery glazing, removal of dust by locally applied exhaust ventilation has accomplished much in the diminution of poisoning. So also has the use of low-solubility glaze or frit, a product in which oxides of lead have been fused with the raw constituents of the glaze, thus converting them into the insoluble lead bisilicate. Up to 1900, red lead or white lead had been almost universally used for pottery glazing, and the good effect of using a non-dusty, highly insoluble substitute can readily be imagined.

In addition to cleanliness in the workplaces, personal cleanliness is of the first importance. Cloak-rooms, washing-rooms, mess-rooms, baths, nail brushes, towels, and soap must be provided. The hands should always be washed before eating, and the workpeople urged to take a warm bath frequently. Food and drink must not be brought into the workrooms and smoking at work must not be allowed.

Medical examination of the workers exposed must be carried out periodically. At present we have no biological test by which to select workmen who are immune to the toxic effects of lead. Since they have been found unduly susceptible, it is necessary to forbid the employment in the potteries and other lead trades of pregnant women and of all persons under eighteen years of age.

The importance of education of the worker himself must be stressed. He must understand fully in what way his work is dangerous. No attempt must be made to hide this or to minimize it, but he must at the same time be shown his own responsibilities in any safety programme. This needs patience and hard work on the part of the doctor. He must inspire employers and workers alike with enthusiasm for safe working conditions. The employer can, and must, do a great deal, but final and complete success

will be impossible without the co-operation of the workman. This co-operation is forthcoming if the approach is the right one; the responsibility for procuring it lies with the doctor.

In the prevention of lead poisoning a diet of high calcium content plays its part. In lead works in Great Britain it has for many years been customary to provide the workmen with a glass of milk each morning free of cost. This is empirical treatment of considerable merit, anticipating as it did by many years the discovery that a high calcium intake assists the storage of lead in a harmless form in the bones. Workers should drink plenty of water which will help to avert constipation and often render the use of aperients unnecessary.

Any worker who shows evidence of abnormal absorption, such as pallor, a drop in the haemoglobin, or a continued rise in the punctate count, must receive treatment. This means stopping lead absorption by suspending him from work in contact with lead and giving him a high calcium diet. Such suspension should not involve financial hardship, and should last for about three months. Any question of return to the old work must receive careful consideration. If it has been possible to modify the operation or conditions of work, including hours, which were responsible for the trouble, the man may be allowed to return. If no such modification is possible, he should be considered unsuitable for that particular job. Workmen should not be allowed to go back to a lead hazard immediately on their return to work after a febrile infection; there is ample evidence that infection is likely to precipitate lead mobilization.

Treatment which aims at the cure or amelioration of lead poisoning should never be made the excuse for negligence in enforcing all the known measures for the prevention of exposure and absorption. Since a high calcium intake causes lead excretion to diminish rapidly, it follows that a high calcium diet is useful in treatment. In mild cases of lead poisoning showing toxic symptoms the diet should contain four pints of milk daily and include milk puddings, junket, and ice-cream, together with butter, cheese, and eggs. Large quantities of calcium lactate, 5 gm. (75 gr.) three times a day, should be given. In the presence of acute symptoms the patient should be admitted to hospital.

In 1948 it was shown that sodium ethylene-diamine tetra-acetate (sodium EDTA; sodium versenate) is a powerful chelating agent having a strong affinity for calcium and heavy metals. This property has been utilized in the treatment of acute and chronic lead poisoning. When calcium di-sodium EDTA (calcium EDTA; calcium versenate) is administered parenterally the lead displaces the calcium and the resulting lead chelate is excreted by the kidney. Not only is the rate of excretion of the metal thus increased, but the circulating un-ionized chelate is much less toxic than would be a similar quantity of ionized metal. Four to eight daily intravenous injections of 3 gm. of calcium EDTA in 600 ml. of 5 per cent dextrose in distilled water are given over a two-hour period. Striking increases in the urinary excretion of lead as high as 13 mg. per day occur.

Treatment of colic with a high calcium diet almost invariably brings relief within two days. The relief of lead colic by calcium therapy involves more than the ability of calcium to favour storage of lead. Since the pain is due to violent peristalsis behind a contracted tonic ring of intestine, the antispasmodic effect of calcium salts on involuntary muscle is beneficial. In severe cases it is possible, by the slow intravenous injection of 15 ml. of a 20 per cent solution of calcium gluconate, or of 10 ml. of a 5 per cent solution of calcium chloride, to relieve the pain by the time the injection is over. The patient feels hot and flushed and may vomit. If necessary, the injection may be repeated in two hours. Should such treatment not be available, a hypodermic injection of atropine sulphate gr. $\frac{1}{60}$ may be given. Enemata of olive oil and mild aperients may be used. Where colic is unrelieved by intravenous calcium gluconate or even morphine, the use of calcium EDTA may be effective.

During the development of *lead palsy* a high calcium diet should be used to favour the storage of lead. Massage and electrical treatment are also useful. In the early stages the hands, when affected by wrist-drop, should be supported on splints.

Lead encephalopathy should be treated by lumbar puncture and a high calcium diet. The control of this condition by the use of calcium EDTA is life-saving.

Poisoning by Mercury

Mercurial poisoning may occur in industry in three forms: (*a*) from exposure to metallic mercury or its vapour; (*b*) from contact of the skin with mercury fulminate, and (*c*) from exposure to organic mercury compounds. The clinical picture is different in each of these three types. Poisoning by metallic mercury leads to stomatitis, erethism, and tremor. Contact with mercury fulminate leads to dermatitis, and methyl and ethyl mercury compounds attack the nervous system, causing severe ataxia, dysarthria, and gross constriction of the visual fields. Only poisoning by mercury itself will be considered here.

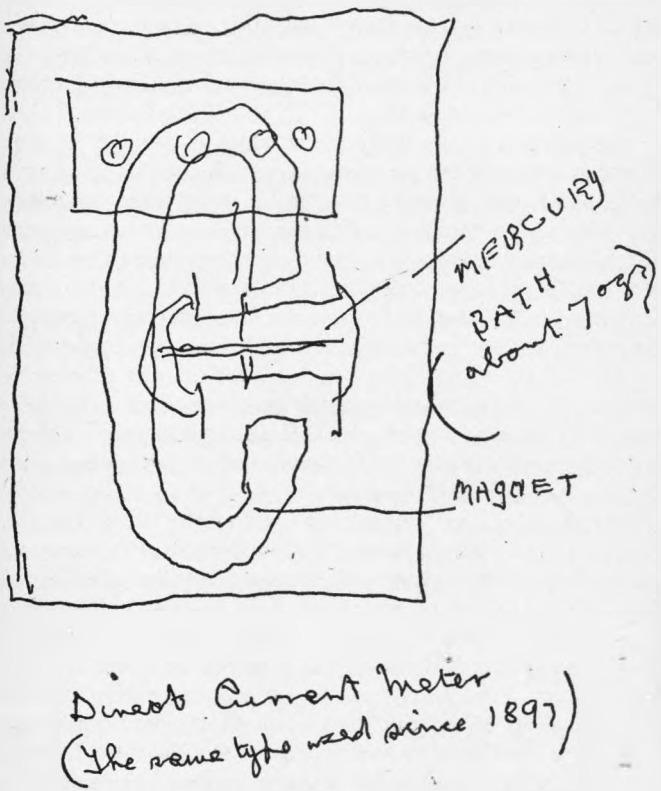
Mercury is a silvery-white metal which is peculiar in being liquid at ordinary temperatures. It was first called quicksilver about 350 B.C. by Aristotle. Mercury is widely used in physical apparatus such as thermometers, barometers, and vacuum pumps. In the electrical industry it is used for mercury-arc rectifiers, contact breakers, automatic switches for refrigerators, and for direct-current meters. The mercury compounds in use include mercury fulminate for use as a detonator, tolyl and phenyl mercury acetate for use as fungicides, and a great number of pharmaceutical compounds and surgical dressings. The sulphide of mercury, vermilion, is indispensable as a red pigment. The red oxide of mercury is used in the manufacture of anti-fouling paints for application to ships' bottoms.

Mercury is a good solvent for some of the metals, and the solutions are called amalgams. Tin amalgam was formerly employed for making mirrors. Gold amalgam is still used for gilding brass buttons. Silver-tin amalgam is used as a dental filling. It is made by mixing mercury with an alloy of silver and tin. When first prepared it is plastic, but after a few hours it sets to a hard mass.

Occupations giving rise to the risk of exposure to metallic mercury include mercury mining, recovery of the metal from the ore, separation of gold and silver from their ores by means of an amalgam with mercury, manufacture of barometers and thermometers, and direct-current electric meters, electric lamps and

radio valves, manufacture of tungsten-molybdenum rod and wire, fire gilding by means of a gold-mercury amalgam, manufacture of pharmaceutical compounds and surgical dressings containing mercury salts, bronzing of field-glasses and photo-engraving, the felting of fur and the manufacture of felt hats, and the identification of fingerprints by dusting with a powder of mercury and chalk.

The *symptoms* of mercury poisoning arising in industry are as



Direct-current meter sketched by a man of 47 suffering from the tremor of chronic mercury poisoning

a rule slower in onset and more insidious in character than those which result from the continued internal administration of mercury. In chronic cases, as, for example, in mercury miners and thermometer makers, two characteristic sets of symptoms which are but rarely seen in medical cases occur, namely tremor and erethism.

Salivation and tenderness of the gums and mouth are usually early symptoms. The gums are swollen and bleed readily, but it is not easy to distinguish an early mercurial gingivitis from the pyorrhoea of a neglected mouth. Rarely a mercurial line is seen on the gums. It usually resembles the blue line due to absorption of lead, but sometimes is dark brown. Mercury may cause *dermatitis* when it is constantly in contact with the skin, as in men who fill the standard 76 lb. iron flasks and spill mercury on their hands and feet. It is a papular erythema with slight hyperkeratosis which affects the dorsum of the hand and foot and spreads to some extent up the leg. With change of work the prognosis is always good.

The most characteristic symptom, though it is seldom the first to appear, is *mercurial tremor*. It is neither so fine nor so regular as that of hyperthyroidism. It may be interrupted every few minutes by coarse jerky movements. It usually begins in the fingers, but the eyelids, lips, and tongue are affected early. It may become less severe when familiar tasks are performed. As it progresses it passes to the arms and then to the legs so that it becomes very difficult for a man to walk about the workshop, and often he has to be guided to his bench. At this stage the condition is so obvious that it is known to the layman as the *hatters' shakes*. The tremor often passes away if the patient gives up his work before it has reached a serious stage. Alcoholism greatly favours its development and it is claimed that no total abstainer has ever suffered from tremor in severe form.

The symptoms known as *erethism* have been rare since silver took the place of mercury in mirror making. The man affected is easily upset and embarrassed, loses all joy of life, and lives in constant fear of being dismissed from his job. Sometimes he is quarrelsome and neglects his work and his family. He has a sense of timidity and may lose self-control before strangers. Thus if a

visitor stops to watch such a man in the factory, he will sometimes throw down his tools in anger and turn on the intruder, saying that he cannot work if watched. Occasionally a man is obliged to give up work because he can no longer take orders without losing his temper, or if he is a foreman because he has no patience with the men under him. Drowsiness by day, depression, loss of memory, and insomnia may occur, but hallucinations, delusions, and mania are now rare. That this has not always been the case may be judged from the expression *as mad as a hatter*.

In 1943 a new physical sign was added to the picture of chronic mercurial poisoning. This was *mercurialentis*, detected by examination with the slit-lamp microscope. The change in the lens consists of a discoloration of the anterior capsule showing as a reflex varying in intensity from light brown to coffee brown. Although the change is bilaterial and symmetrical, visual acuity is unaffected. The discoloration of the lens capsule appears a long time before the onset of general signs of mercury poisoning and is therefore of value in the early detection of exposure to atmospheric mercury.

The work of chemists leading up to the discovery of *safe substitutes* for mercury dates back more than a hundred years. Mercury poisoning was common in the mirror-making industry until silvering was introduced. In 1835 Justus von Liebig observed that a test tube becomes coated with silver if an ammoniacal solution of silver nitrate is warmed with an aldehyde. The mirror so formed provided a more perfect reflecting surface than one made with tin amalgam, and mercury poisoning disappeared in the trade. Similarly powders containing mercury or its compounds used for the identification of fingerprints must also disappear. Salts of calcium, bismuth, barium, zinc, or titanium should be used instead.

The introduction into commerce of electrolytic gold plating in 1840 also led to a great reduction of mercury poisoning because this process largely replaced amalgam plating. *Protective measures* in mining must include good ventilation, wet methods in drilling, and the use of canister respirators containing both iodized carbon and activated charcoal. Periodical medical examination should aim at the frequent alternation of workers at danger points and

should pick out all men showing any symptoms of mercury poisoning. These must either be withdrawn immediately or given other jobs presenting no mercury hazard. In smelting plants, especially dangerous operations are the charging of furnaces, the processing of soot containing up to 80 per cent of metallic mercury, the filling of iron bottles, and the cleaning of condensation pipes and chimneys. Men doing these jobs must wear respirators or hose masks. Measures for general hygiene should include washing facilities, shower baths, and lockers.

The mistake of forcibly ventilating open workshops must be avoided, since it increases the vaporization of mercury. Exhausts should be placed to draw off the air from apparatus where mercury is used, and the work space supplied with fresh air from an outside source. Concentrations of mercury should never rise above 75 micrograms per cubic metre whatever the weather conditions. Where fire gilding is employed for gold plating of metal objects, adequate ventilation must be provided. A water trap must be built into the flue of the furnace used in order that the mercury vapour driven off will condense, collect under the water, and remain harmless.

In the furriers' workshops of the hat trade the technical processes of carroting, drying, brushing, sorting, and packing are carried out; if the fur-cutting shops are small, cheaply built, and badly managed, poisoning will readily arise. Exhaust ventilation and spotless cleanliness must be introduced in such shops. These measures may not eliminate all risk, for after the carroted fur has left the furriers' workshops it goes through further processes known as blowing, forming, hardening, sizing, blocking, shaping, crown and brim ironing, planking, proofing, stoving, and pressing. In Great Britain preventive measures have reduced the number of cases of poisoning to negligible proportions.

In all processes in which mercury is handled it is necessary to observe strict cleanliness. Mercury can seep into crevices, collect in interstices, and penetrate wooden floors. Since it vaporizes at room temperature, harmful concentrations may occur in the atmosphere from what may seem negligible spillage. Construction in wood should be avoided; floors of concrete should be maintained without cracks or open interstices. More attention should

be paid to the risk of poisoning in the filling, emptying, and repairing of apparatus such as vacuum pumps, mercury rectifiers, and electro-medical apparatus. Here it is difficult to prevent drops of mercury from falling on the floor, and a safety device incorporating a water trap must be built into the floor of the workshop. This consists of a large flat trough full of water beneath the working-point covered by an iron grill. When mercury is spilt on the floor it falls through the grill into the trough containing water. The mercury is then easily removed and purified for further use. Benches should be covered with a smooth and impervious surface tilted in such a way that mercury can be drained and collected, thus preventing vaporization and contact with the skin. Wherever possible, mercury should be handled in enclosed apparatus.

Overalls, mess-rooms, and adequate washing facilities must be provided, and there must be strict supervision to ensure that the workers wash their hands after each shift and before all meals. The mouth and pharynx should be frequently rinsed with a mouth-wash and the teeth cleaned with a soft tooth-brush and a dentifrice. *Periodical medical and dental examination* can achieve a great deal, especially by emphasis on the proper hygiene of the mouth. Cavities in carious teeth should be filled, sharp angles smoothed, and useless teeth extracted.

In cases of acute mercury poisoning, where mercury perchloride or some other soluble compound of mercury has been swallowed by accident or for purposes of suicide or homicide, therapy with 2:3-dimercaptopropanol (British Anti-Lewisite or BAL) if used in time is life-saving. It is therefore disappointing that the slow absorption of mercury vapour in the victims of industrial poisoning denies them the benefit of this treatment. Indeed, it is not yet clear whether in chronic mercury poisoning the mercury-BAL complex may not be more harmful than the mercury itself.

Poisoning by Arsenic

Arsenical poisoning may occur in industry in three forms: (*a*) from inhalation of or contact with the dusts of inorganic

compounds of arsenic; (*b*) from inhalation of arseniuretted hydrogen gas; and (*c*) from contact with organic arsenic compounds. The symptoms and signs in the three groups are distinct. The inorganic compounds of arsenic act as local irritants to the skin and mucous membranes, and may have a carcinogenic effect. Arseniuretted hydrogen acts as a haemolytic agent, causing haemoglobinuria, anaemia, and haemolytic icterus. The organic arsenic compounds have a vesicant effect on the skin and the mucous membranes as well as powerful systemic effects. Only poisoning by inorganic arsenic compounds will be considered here.

In industry, arsenical compounds are met with in the smelting and refining of ores, in the subliming of white arsenic, and in the manufacture of insecticides and weed killers. White arsenic is used as a preservative of hides, skins, furs, and wood. In the manufacture of glass it is used to remove the unpleasant greenish tint produced by iron oxide. Cupric aceto-arsenite is used as a horticultural spray to kill the codling moth and the gipsy moth on fruit trees. Lead arsenate spray or dust is used to kill the cotton-boll weevil, and cupric arsenite to kill the potato bug. Other insecticidal sprays and dusts contain calcium arsenite and arsenate, magnesium arsenate, manganese arsenate, and zinc arsenite. Sheep dips, cattle dips, and powders to kill flies and ants may contain sodium or potassium arsenite, arsenious oxide, arsenic sulphides, and thioarsenates. Arsenic compounds are used in anti-fouling paints for ships' bottoms, in calico printing, and in the manufacture of pharmaceutical substances. About 0·4 per cent of arsenic added to molten lead yields harder lead shot and facilitates also the formation of truly spherical pellets when the melt is poured down the shot tower.

The dusts of the arsenical compounds manufactured in industry are light, so that, unless the processes of sifting and packing are carried out in closed apparatus from start to finish, the dust is likely to alight on the skin and remain there. The skin is affected especially where there are folds, as around the nose and mouth, or around the edges of a respirator, or where surfaces are moist, as in the axillae or on the scrotum.

A dermatitis is set up in these areas. It consists of eczema,

sometimes with oedema and the formation of bullae, and folliculitis in varying degree. If untreated it leads to ulceration, which may be extensive. Associated with the skin eruption are conjunctivitis, with oedema of the eyelids, coryza, dryness of the throat, and hoarseness. In severe cases vomiting occurs, but colic is rare. Headache and paraesthesiae in the limbs may occur, but widespread polyneuritis is rare and motor involvement is practically never seen. Finely mottled brown pigmentation of the skin, the so-called raindrop pigmentation, usually on the temples, eyelids, and neck, is present in those who have worked for years in contact with arsenical dusts. In severe cases there may be intense bronzing of the chest, abdomen, and back. The most characteristic lesion produced in the upper air-passages is perforation of the nasal septum, which is painless and may be complete in a month from the time of starting work. On the other hand, by the faithful use of a dust mask men have worked for years without developing such a lesion. Once the perforation is complete there is no further extension of the erosion and the worker may be unaware of the existence of the condition.

The fine powder of arsenical compounds which settles on the skin of the industrial worker may give rise to warts on the nostrils, eyelids, lips, ears, and wrinkles of the neck, and, since these compounds of arsenic are carcinogenic, the warts may become malignant. Cases of cancer of the skin due to occupational exposure to arsenic occur from time to time, but they are rare. The clinical picture is made up of pigmentation, keratosis, and single or multiple squamous carcinomata. There are no special sites; the face, abdomen, scrotum, buttocks, clavicle, and chest wall may be affected.

In the *prevention* of arsenical poisoning in industry, dust must be suppressed. The floors of workrooms and passage-ways should be of impermeable material, and they should be frequently flushed with water. Workrooms should be well ventilated and hoods connected with a good draught placed over apparatus emitting dust. All poisonous fume should be condensed and any dust caught removed. Hot processes should be carried out under glass hoods and manipulation of powders in closed glass cabinets. When possible, mechanical methods should take the place of

hand labour. Apparatus and receptacles must be strong, to avoid breakage. In all processes in which arsenical dust is likely to arise, tables should be provided with downward exhaust ventilation. Since the dust of cupric arsenite is light, it is difficult to protect the workers handling it. Automatic packing is not possible, and the ordinary means of protection by respirators favours sweating and consequent ulceration of the skin. Persons with moist skin and those who sweat readily are unsuitable for the work and should be excluded. Air-line helmets, protective overalls, washing accommodation, and towels should be provided (Plate 4). Neither food nor drink should be taken in the workroom, and smoking and the taking of snuff should be prohibited.

In *treatment* deep intramuscular injections of 300 mg. of 10 per cent oily suspension of 2:3-dimercaptopropanol (BAL) repeated every six hours have a specific effect upon the dermatitis, conjunctivitis, and pharyngitis of workers in arsenic compounds. Itching, pain, and swelling begin to disappear after the first dose. Subsequently, daily injections of 150 mg. should be given for two or three days.

Poisoning by Manganese

Manganese when pure is a silvery-white metal, but as usually prepared it is reddish-grey, brittle, and intensely hard. About 95 per cent of the world production of manganese is used for metallurgical purposes, particularly the manufacture of manganese-alloy steels. The metal industry could not dispense with it and uses an average of 14 kilograms of manganese per ton of ordinary steel. Its most important alloys are ferro-manganese, spiegeleisen, silico-manganese, silicospiegel, and manganese-bronze. During the process of steel making, ferro-manganese is added to the furnace charge to prevent the formation of iron oxide and sulphide in the finished steel. A small amount of manganese in steel increases its elastic limit and tenacity, so that steels containing about 1 per cent of manganese are commonly used in structural work. For rock-crushers, dredger buckets, railway points and crossings, clutches, steel helmets, and in certain mining equipment requiring high-tensile strength and resistance to shock and

abrasion, high-manganese steel with about 12 per cent of manganese is employed. At Baker Street station on the London Underground Railway ordinary steel rails at the crossings used to need replacement every 9 months, but the manganese steel now used lasts 22 years. Alloys are also made with aluminium, tin, arsenic, antimony, bismuth, and boron.

The dioxide is the starting-point in the manufacture of all manganese preparations. It is used in decolorizing glass stained by traces of iron compounds, for the violet colour of manganese silicate masks the complementary green tint of the iron. The pyrolusite used for this purpose is known as *glass makers' soap*. The dioxide is much used in the manufacture of dry batteries and in the pottery and soap industries. Manganous chloride is used in dyeing; and manganous sulphate in calico printing. The insoluble colours formed in the dye vats are referred to variously as *manganese brown*, *manganese bistre*, and *manganese violet*. Manganates and permanganates, especially potassium permanganate, are used for preserving wood, for bleaching textiles, and for oxidizing and disinfecting purposes. *Condy's fluid* is a mixture of sodium manganate and permanganate. Although manganese dioxide is still used to oxidize hydrochloric acid to chlorine on a laboratory scale, this process is obsolete in industry, the electrolysis of brine having replaced it.

Cases of manganese poisoning have been seen from the inhalation of excessive amounts of dust in the mining, grinding, sorting, sieving, packing, and loading of manganese ores and in the manufacture of manganese steel in which the manganese is first fused in an electric furnace. In Great Britain no case has been recognized in the manufacture of dry batteries. Of the number of persons exposed, few are susceptible to the disease. Manganese poisoning produces two entirely different effects: the first is an attack on the brain with strict localization to the extrapyramidal motor system, a condition discovered in France in 1837; and the second an increased incidence of pneumonia first noted in Germany in 1921.

The symptoms and signs include languor and sleepiness by day but insomnia by night, muscular pains, including cramps in the calves, unsteady gait, weakness and stiffness of the limbs, and involuntary movements varying in degree from a fine Parkin-

sonian tremor of the hands to gross rhythmical, rotatory movements of the arms, legs, trunk, and head which may be severe enough to shake the bed. There may be propulsion, retropulsion, mask-like facies, and sialorrhoea. Occasionally uncontrollable laughter or crying occurs, and there may be impulsive acts such as running, dancing, singing, and uncontrolled talking. Sometimes forced movements occur in which the patient falls without being able to make the effort necessary to save himself. Attacks of aggressiveness, unprovoked irritability, and euphoria are known. The handwriting is tremulous, the letters and words cramped, and micrographia is common. Speech disturbances include disappearance of the pauses between words, monotonous tone of voice, and in severe cases aphonia. Occasionally deglutition is impaired. In Cuban mines drilling and blasting an ore containing 45 per cent of manganese, impotence is one of the commonest manifestations of poisoning.

Although men seriously poisoned are lifelong cripples, the condition is not lethal. The emotional alterations are usually transient only; the extra-pyramidal symptoms and signs persist. In any group of workmen showing neurological symptoms and signs it is unusual for as many as 10 per cent to recover sufficiently to resume work. In the remainder the weakness, spasticity, and tremor render impossible any return to the former employment.

In 1921 attention was first drawn to the unusually high incidence of pneumonia in men handling manganese ores. In 1933 the same thing was found amongst dry battery workers handling manganese dioxide and in 1946 in men employed in the manufacture of potassium permanganate. In manganese pneumonitis the response to sulphonamides and resolution of the lung are slower than in ordinary lobar pneumonia, but no persisting pulmonary lesions are observed either clinically or radiographically.

In the mining of manganese ores adequate ventilation and wet rock drilling are essential precautions underground. Baths and changing rooms must be provided on the surface. In factories manganese poisoning can be prevented by the application of local exhaust ventilation, both at the furnaces to remove fume and at the packing and sieving apparatus to remove dust. Respirators may be worn which combine active charcoal for absorbing

vapours and a cotton-wool filter to trap dust. Personal hygiene is important, and the worker must wear protective clothing and gloves, since skin absorption is known to occur. Adequate supervision and routine medical examination are essential. These measures are attended with good results.

No effective method to increase manganese excretion is known. Both 2:3-dimercaptopropanol (British Anti-Lewisite, BAL) and calcium EDTA are without effect. Symptomatic treatment includes simple anodynes for muscular pain and barbiturates for insomnia. For the rigidity, gentle exercise, passive movements, and massage are useful. Some relief may be obtained by hyoscine hydrobromide given three times a day as a tablet of gr. $\frac{1}{100}$. This usually renders movements more free and relieves the tremor for a few hours after each dose is taken. Patients often become bedridden and therefore need institutional treatment. It is then necessary to take great care of the skin, since the immobility of the trunk increases the liability to the formation of bed-sores.

Poisoning by Nickel

Exposure to nickel compounds creates three distinctly different hazards: (*a*) contact with solutions of nickel salts in refining the metal and in electroplating may give rise to dermatitis; (*b*) inhalation of nickel carbonyl, a gas which is highly poisonous and may cause death from haemorrhagic bronchopneumonia; and (*c*) inhalation of dust in the refining of nickel, giving rise to cancer of the lung and nasal sinuses, especially the ethmoid sinus. Only the effects of contact with nickel salts will be considered here.

Nickel was first isolated in 1751. It is a hard, silver-white, corrosion-resistant metal which takes a high polish. It is malleable, ductile, and very tenacious. Pure nickel is used principally in electroplating, either as the complete coating or as an undercoat to a chromium finish. It forms an unusually large number of alloys of technical importance. Nickel is a metal similar to iron in some of its properties, having rather greater strength and hardness and being magnetic, though to a smaller degree than iron. Ferro-nickel alloys have a wide range of magnetic, electrical, and

thermal properties, depending on the percentage of nickel used. Thus *invar*, with 36 per cent nickel, has the remarkable property of not expanding or contracting appreciably with ordinary changes of temperature. It gets its name because it is invariable at all air temperatures. It is useful for standards of length, measuring tapes, and chronometer parts. *Platinite*, with 46 per cent nickel, has the same coefficient of expansion as glass and can therefore be used as a substitute for platinum for the lead-in wires of electric-light bulbs. Nickel is used in stainless steels, and to add strength and toughness to steel alloys for motor cars, aircraft, gun forgings, armour plate, and machine tools, including cemented-carbide cutting tools. It is alloyed with copper and other metals to make condenser tubes and coins. Together with zinc and copper it forms *nickel silver*, the basis of plated tableware. *Monel metal* is an alloy of nickel with copper and small quantities of iron, manganese, silicon, and carbon. It is malleable and of high tensile strength, and is used for kitchen ware, laundry fittings, food-handling plant, pump bodies, and turbine blading. Of the alloys acting as permanent magnets, *alcomax* is composed of nickel, iron, and aluminium, and *alnico* of nickel, cobalt, and aluminium. Alloyed with chromium, it is used to make heating elements for electric fires and surgical and dental instruments. In the Second World War modifications of the binary alloy nickel 80, chromium 20, were developed to withstand the combination of high stresses and high temperatures met with in gas-turbine engines and at the same time to resist corrosion by the products of combustion. They are known as the *Nimonic series*. Finely powdered nickel is used as a catalyst in a number of chemical reactions, especially in the hydrogenation of oils to form solid fats. Nickel and its salts are used in alkaline storage batteries and in the manufacture of enamels.

In 1908 graphic descriptions were written of the *nickel itch* which occurs in nickel-plating establishments. It also affects nickel miners, smelters, and refiners. It begins as a pink, papular erythema in the web of the fingers. There is itching, soreness, burning, and sometimes swelling of the parts affected. The itching is worse at night and in hot weather. The eruption may spread on to the fingers, wrists, and forearms, and at times on to the chest

and face. In extreme cases pustulation, ulceration, and weeping occur. As a rule recovery takes place after a week, although cases are known where the condition has lasted for three months.

Rarely, the patient complains of a bitter metallic taste in the mouth, with loss of energy both physical and mental. There is a heavy preponderance among fair-skinned employees. Some persons are extraordinarily susceptible to nickel salts and others become sensitized only after having worked as electroplaters for years. One man in three seems to be immune. If a man has once been affected, he is almost sure to have another attack in which the lesions are slower in healing than in the first. Such a man may be compelled to abandon the occupation for good.

Persons who are sensitive or show previous susceptibility to nickel dermatitis should be excluded from working on electroplating vats. Discipline and regular medical supervision should ensure absolute cleanliness on the part of the workmen. Cuts and abrasions must be covered by suitable dressings. The exposed skin should be washed, carefully dried, and treated with an ointment of lanolin and soft paraffin. If the workmen wear gloves, boots, and aprons, they should understand how to use them properly. Removal of dust and mist from the atmosphere is essential and depends on the correct design of the vats to incorporate exhaust ventilation.

Poisoning by Chromium

Chromium is a silver-white, hard, brittle metal. Approximately 45 per cent of the world's supply is used for alloys, about 40 per cent for refractories, and 15 per cent for chemical purposes. Chromium is an essential component of the hard, high-strength steels used in engineering, and also of stainless and rustless steels. Corrosion-resistant alloys, such as stainless chromium-nickel steel, are extensively used in the chemical industry. The alloy steels are manufactured directly from ferro-chrome, a chromium-iron alloy prepared by reducing chromite in an electric furnace in the presence of carbon. Nickel-chromium alloys are used for making resistance wires for electric fires, copper-chromium alloys for electrical switch gear, and cobalt-chromium-molybdenum alloys

for use in gas-turbine engines and in orthopaedic surgery. For refractory purposes the crude ore is embodied in bricks, cements, and plasters in the construction of furnaces. The only commercial use of pure chromium metal is in the form of electroplate. The chromates of lead, zinc, and barium are known as chrome pigments and are extensively used for colouring paints, linoleum, rubber, and ceramics. Chromium sulphate is important in tanning, and potassium dichromate in the dyeing of wool, silk, and leather. Chromium compounds are used in photography, in making safety matches, and as catalysts in the manufacture of aviation petrol and methanol.

Air contaminated with chromic-acid mist or with the dust from chromates or bichromates is the principal source of exposure in industry. The process of chromium plating consists in wiring the articles to a frame ready for the plating vat, the initial plating, which lasts up to fifteen minutes, and the unwiring, swilling, and polishing of the plated articles. The solution in the plating tank contains 50 per cent of chromic acid, and during electrolysis reddish-brown fumes which contain 60 per cent of chromic acid are forced up in the form of a mist by the evolution of hydrogen at the cathode. In anodizing, a coating highly resistant to corrosion is formed on aluminium and its alloys through the anodic oxidation of the aluminium. Chromic acid is used as the solution in which the anodizing operations are carried out, and the hydrogen liberated at the cathode carries a significant amount of chromic-acid mist into the atmosphere along with it.

Lesions of the skin due to chromium salts have been known since 1827, when *chrome holes* were described on the fingers and hands of bichromate workers in Glasgow. The chromates and bichromates of potassium and sodium and chromic acid may cause either dermatitis or localized ulceration, according to whether trauma is present or not. Exposure to these substances occurs in chromium platers, colour workers, French polishers, calico printers, photographers, litho-etchers, and chrome tanners. Dermatitis may occur on the hands, arms, face, and chest. The onset is sudden, but it is unusual for an attack to occur until the operator has been at the work for at least six months. In severe cases the face is intensely red and swollen, and the affected parts

itch a great deal and may become painful. Fair-haired people are particularly prone to a chrome dermatitis, and their presence at a chromium-plating bath calls for peculiar care.

Chrome ulcers begin in abrasions of the skin and are most commonly found at the root of the finger-nail, the knuckle of the hand, or the dorsum of the foot. They are circular in shape, clear-cut, usually one centimetre or less in diameter, and punched out, hence the name *chrome hole* (Plate 18). They have a strong tendency to heal but may penetrate very deeply, even to bone. Although painless, they itch intolerably at night. If neglected, an ulcer may give rise to infection at the adjacent joint, causing loss of a finger. There is no tendency towards malignant change. The dust of chromium salts and the mist of chromic acid may produce ulcers on the eyelids or the edge of the nostrils.

The mucous membrane of the nose is commonly affected, in which case *perforation of the nasal septum* occurs. Usually this causes no inconvenience and is discovered accidentally. The condition appears between the sixth and twelfth months after beginning work. The site of election for the ulceration is a point about a quarter of an inch from the lower and anterior margin of the septum, and from this point it extends upwards and backwards. The limitation of the perforation to the cartilage of the septum is accounted for by the fact that the mucous membrane covering it is adherent, forming the perichondrium, and is far less vascular than the mucous membrane lining the rest of the nasal fossa. Once the mucous membrane is destroyed, the blood supply to the cartilage is cut off and necrosis ensues. When the ulceration has progressed upwards as far as the junction of the septum with the ethmoid and backwards to the vomer, it becomes arrested. Healing then takes place without the bone being attacked, and the scar usually becomes covered with a crust of mucus. Since the anterior or lower border of the septum is never destroyed, the rigidity of the parts is maintained and deformity does not occur. The onset of the process is ushered in by sneezing and by the symptoms of nasal catarrh. The pain accompanying the ulceration appears to be insignificant. It is never severe enough to necessitate absence from work or to call for treatment. Once the perforation is established, the only inconvenience is the

formation of plugs of mucus in the nasal passages. The general health is unaffected by the condition.

The *preventive measures* necessary in handling chromic acid, chromates, and bichromates include the removal of dust and mist, cleanliness, regular medical supervision, and the covering up of cuts and abrasions with suitable dressings. The prevention of danger in chromium plating and anodizing depends on the correct design of the vats to include exhaust ventilation. It may often be necessary to provide rubber gloves, boots, and apron for a person working round the chromic-acid tanks, but these are not always of real value. The workers have to reach up to lift out the metal parts and hang them on hooks, and if the gloves are loose at the wrists the solution that drips on to the arms may, when the hands are lowered, run down inside the gloves. The solution may also run down inside the boots if they are loose at the top. The exposed skin should be washed and carefully dried. Application of an ointment made up of equal parts of lanolin and soft paraffin is useful. Soft paraffin should be freely applied through the anterior nares to the nasal septum. Weekly inspection of the nasal passages should be regularly carried out for all workers by the nurse, and less frequent inspection should be made by the works doctor. Chrome ulcers can be successfully treated by an ointment containing 10 per cent edathamil calcium (calcium EDTA).

Poisoning by Beryllium

Beryllium is a very light, hard, silvery-white metal. It is used in the preparation of beryllium-copper alloys, in the generation of atomic energy, in the manufacture of radio valves, and as a refractory in the manufacture of crucibles and electrical porcelain. Its use as a constituent of phosphors for fluorescent lamp tubes and neon signs has been discarded. Beryllium metal, beryllium oxide, and some beryllium salts can act as irritants in contact with the skin and mucous membranes. Inhalation of the dust of the metal or of zinc beryllium manganese silicate, at one time used for phosphors, tends to cause chronic granulomatous lesions especially in the lungs. The respiratory illness may not become apparent until several months or even years after exposure has

ceased. Individual susceptibility to poisoning by beryllium and its compounds varies within wide limits.

Continued exposure of the skin to dust or fume of beryllium or its salts, especially when accompanied by perspiration and friction, is liable to cause an oedematous, papulo-vesicular *dermatitis* usually localized to the part exposed and generally clearing up rapidly on cessation of exposure. *Conjunctivitis*, *rhinitis*, *bronchitis*, and *pneumonitis* have been found to follow even short exposures to beryllium-bearing dust or fume arising from the metal, oxide, sulphate, chloride, and fluoride. The *naso-pharyngitis* and *tracheo-bronchitis* are characterized by slight fever, unproductive cough, and dyspnoea. Pneumonitis starts insidiously with slight fever, cough, retro-sternal pain, dyspnoea, and scanty sputum. The pulse tends to be disproportionately rapid, râles appear over both lungs, and the vital capacity is much reduced. Several weeks after the onset, x-ray examination may reveal a diffuse haziness thoughout both lungs.

Inhalation of zinc beryllium manganese silicate in a state of fine subdivision has resulted in many cases of *delayed pneumonitis* or *chronic berylliosis*. The delayed onset is a striking feature, the period between cessation of exposure and the appearance of symptoms varying from a few months to 5 years or more. In these cases, too, the onset is as insidious as it is delayed. Patients seek advice because of loss of weight and fatigue. The loss of weight is progressive and is followed by gradually increasing dyspnoea. Cough and expectoration are not prominent features at any time. Progressive dyspnoea becomes distressing and ultimately is apparent at rest. Physical signs in the chest are not pronounced; there may be scattered adventitious sounds. In early cases x-rays of the lungs show a slight generalized haziness, but later reticular shadows appear and coalesce slowly, assuming the picture of widespread discrete nodulation.

Deep implantation of certain beryllium salts in wounds, especially those resulting from the breakage of fluorescent lamp tubes, may be followed, after an interval, by the development of a *local benign granulomatous nodule in the skin*. Similar lesions may be found at necropsy in the liver, glands, and other organs in cases of chronic berylliosis.

The *prognosis* of beryllium dermatitis is that it readily clears up and leaves no residual increased sensitivity. Acute inflammation of the upper respiratory tract is usually of short duration, but an acute pneumonitis may remain active for several months. Such pneumonitis tends to progress, without exacerbations or remissions, either to complete recovery or death, the mortality rate being in the region of ten per cent. About a third of the cases of delayed pneumonitis are fatal, while a further third are permanently disabled.

Because the poisonous nature of beryllium and its compounds has been known only since 1933, there is much room still for education of those who handle it. At a time when cases of beryllium poisoning in the United States fluorescent-lamp industry were occurring with greatest frequency, the phosphors contained beryllium in percentages ranging from 4 to 12. Later, the figure was reduced to 2 per cent, and now halo-phosphates containing no beryllium are available as safe substitutes for the poisonous phosphors of the past. Since chronic berylliosis has occurred in smelters of beryllium and copper and in others who machine the finished alloy, it is desirable that safe substitutes be developed. An alloy containing 60 per cent copper, 20 per cent nickel, and 20 per cent manganese has properties similar to those of beryllium-copper and for certain purposes can be used in its place. In Great Britain in 1949 poisoning by beryllium and its compounds was added to the list of diseases prescribed under the *National Insurance* (*Industrial Injuries*) *Act*.

Preventive treatment consists in the enforcement of strict measures for dust suppression in all industries where beryllium and its compounds are used (Plate 5). There is evidence that cases of disease will occur when exposure to beryllium as oxide exceeds 100 micrograms per cubic metre of air. Every effort must be made by engineering methods to keep the atmosphere concentration in the working environment lower than 2 micrograms per cubic metre of air. Protective clothing and adequate laundry services should be provided. No factory effluent must be allowed to endanger the health of people living in the neighbourhood.

Workers at risk should be examined at regular intervals by a doctor and questioned about suggestive symptoms. Accurate case

records should be kept. All persons exposed must be weighed at monthly intervals and have the chest x-rayed at least once a year. Repeated medical and x-ray examinations must be made of all workers who have unexplained symptoms, particularly when these suggest disease of the respiratory system.

In the *treatment* of acute pneumonitis patients must be kept for many weeks in bed. Before the serious nature of the disease was appreciated, several patients died from a relapse of pulmonary symptoms and signs which occurred on getting them up too soon. Treatment is symptomatic. The patient should be propped up with a canvas bed-rest and sedatives should be given for the cough. In those severely affected, oxygen is necessary to relieve the dyspnoea. Penicillin-streptomycin aerosol therapy has relieved secondary bacterial infection but has no other beneficial effect. Prolonged convalescence up to 4 or 6 months is necessary; it must continue until x-rays of the chest show no abnormality.

In the chronic disease, certain patients, although losing weight and suffering from mild dyspnoea, are capable of some type of work, perhaps only part time. Since subcutaneous granulomata have developed in persons who have cut themselves on broken lamps, caution must be exercised in the disposal and salvage of burnt-out fluorescent tubes. It is best to break them under water and to bury the fragments in the ground.

Poisoning by Cadmium

Cadmium is derived from zinc-bearing ores. It is used as a constituent of alloys, solders, bearing metals, and the negative plates of alkaline storage batteries. It is also used for the rust-proofing of iron and steel articles, a thin film being applied either by electrolytic deposition or directly by pressure spraying of cadmium metal drawn into wire. Poisoning may occur when fume from molten cadmium is encountered as, for example, in smelting processes, metal pouring, the manufacture of copper-cadmium alloys, spraying of atomized metallic cadmium on a base metal, and welding of cadmium-plated articles. Freshly produced cadmium fume is highly irritating to the mucous membranes of

the eyes, nose, and throat. In low concentrations such fume is respirable and may act as a cumulative poison.

Acute poisoning may occur from brief exposure to a high concentration of cadmium fume. The symptoms are smarting of the eyes and nose, dryness and irritation of the throat, tightness of the chest, dyspnoea and cough, going on, if exposure is more intense, to nausea, retching, vomiting, and marked prostration. Later there may be an attack of metal-fume fever with rigors and pyrexia. In a severe case these symptoms are followed rapidly by acute pulmonary oedema which, if severe, may cause death from anoxia. If it does not, it is followed by a proliferative pneumonitis which may be fatal or may resolve completely.

Chronic poisoning may occur in the form of chronic emphysema. In this type of case the early symptoms may be vague. Some men give a history of continued nasal and pharyngeal irritation, perhaps with recurring epistaxis and, in some cases, anosmia, but more commonly the complaint is of a more general nature namely, undue tiredness, dyspnoea, and cough. Clinical and radiological examination of these men may show emphysema unconnected with bronchitis or asthma. This would appear to be a local effect, a result of repeated exposure to recently generated cadmium fume. Cadmium is excreted through the kidneys, and its continued presence in the urine after exposure has ceased may be regarded as evidence of accumulation. Associated with the renal excretion of cadmium there is frequently excretion of an unusual protein of molecular weight as low as 20,000 or 30,000. This protein may escape detection unless trichloracetic acid or sulphosalicylic acid is used as a precipitant. Its presence in the urine lends support to a diagnosis of chronic cadmium poisoning where a cadmium worker suffers from emphysema otherwise unexplained.

After an acute attack of cadmium poisoning, the result of exposure to fume, recovery is to be expected and is likely to be complete. Chronic poisoning is not likely to attract attention until it has resulted in a clinically recognizable degree of emphysema. This leads ultimately to right heart failure. Death from renal insufficiency is rare.

In *preventive treatment* it must be remembered that whenever

cadmium is heated, dangerous quantities of cadmium oxide are formed and volatilized. Therefore in the smelting of cadmium ores, the manufacture and welding of alloys, and the firing of cadmium-plated metal, precautions should be taken to remove all fume by means of adequate exhaust ventilation (Plate 6). It has been suggested that all cadmium-coated metal should bear a warning label. While this measure is effective for large pieces, it is somewhat difficult to ensure that small objects so coated are labelled. Symptomatic treatment is directed specifically against pneumonia when it occurs.

Poisoning by Vanadium

Vanadium was discovered in 1830. The pure metal is difficult to obtain even on a small scale on account of the high temperature necessary and the tendency to re-oxidation. It is important to note that in addition to its occurrence as vanadium ores it is also present in certain terrestrial plants, in sea water, in marine muds, and in certain shales and crude petroleum oils. Vanadium occurs regularly in the blood of certain *Ascidiacea*, or sea squirts, and *Holothuroidea*, or sea cucumbers, where it forms up to 10 per cent of the blood cell pigment. These creatures live fixed to rocks and their fossilized remains account for the presence of vanadium in petroleum found in certain parts of the world. The percentage of vanadium in the ash of crude oils varies a good deal. Thus in samples from Venezuela it may be as high as 45 per cent, Oklahoma 22 per cent, Iran 14 per cent, and California 5 per cent. More than 20 tons of vanadium pentoxide are recovered annually from soot which collects in the boilers and smoke-stacks of ships burning Venezuelan and Mexican fuel oil.

About 95 per cent of the world's supply of vanadium is consumed in the manufacture of special alloy steels. In the form of ferro-vanadium it is added to steel to promote fineness of grain, toughness, and resistance to torsion and high temperature. The tensile strength of steel is raised from 7·5 to 13 tons per square inch by the addition of 0·5 per cent of vanadium. This property commends itself in the manufacture of forgings for locomotive and motor-vehicle parts, including transmission shafts, gears,

axles, and springs. Vanadium pentoxide is used as a catalyst in the oxidation of naphthalene and is replacing platinized asbestos in the contact process for the manufacture of sulphuric acid.

In 1911 vanadium poisoning was described in a factory where vanadium ore was ground. Ill effects of exposure to vanadium compounds in the cleaning of oil-fired boilers were first recorded in 1952. The workmen concerned entered the combustion chambers and dislodged the fine soot from the brick-lined walls and from the heating tubes.

The *symptoms and signs* include smarting and irritation of the eyes, shortness of breath, pain in the chest, palpitation on exertion, and paroxysmal cough with profuse expectoration and rarely haemoptysis. There is greenish-black discoloration of the tongue, conjunctivitis often with suppuration, tremor of the fingers and arms, and rhonchi throughout both lungs. In severe cases death occurs from broncho-pneumonia.

In order *to prevent poisoning* by vanadium compounds in industry, mechanization and enclosure of all dusty processes must be strictly enforced. In boiler cleaning the operator must stand outside the boiler using a long compressed-air lance. The soot must be drawn away from him into the flue by an induced draught fan. In cases where certain parts of a boiler cannot be reached by this method, men entering the boiler must wear protective clothing and suitable dust masks. All men exposed to vanadium compounds must be under medical supervision and must be re-examined periodically. Pre-employment examination should include radiographic examination of the chest and a patch test of the skin with a 2 per cent solution of sodium vanadate.

CHAPTER SIX

POISONING BY ORGANIC COMPOUNDS

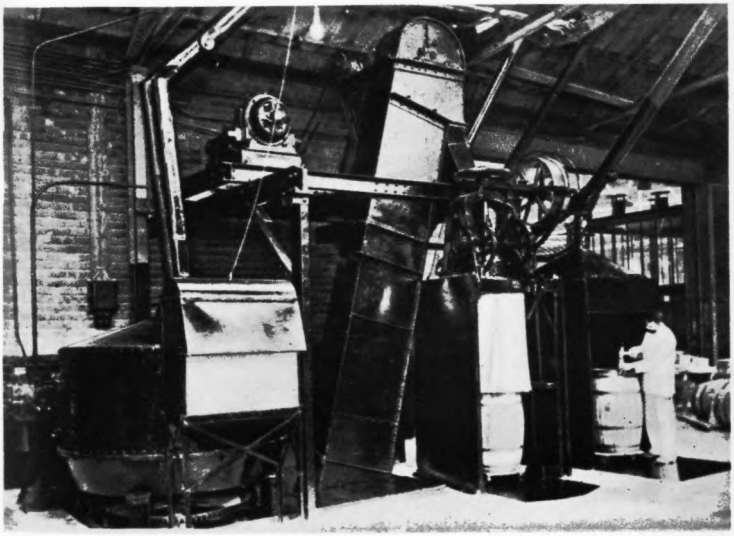
Coal-tar Derivatives– Halogenated Hydrocarbons – Tetraethyl Lead – Organic Mercury Compounds – Organic Arsenic Compounds – Organic Phosphorus Insecticides

EARLY in the nineteenth century the French chemist Gay-Lussac and the Swede Berzelius opened a new era, that of organic chemistry, which revealed the structure of the carbon compounds associated with the living processes taking place in plants and animals. In Gay-Lussac's private laboratory, the young Liebig collaborated with his chief in researches on the salts of fulminic acid, and in 1824 the composition of this acid was established. Meanwhile Wöhler was working in Stockholm with Berzelius and almost at the same time determined the composition of cyanic acid. The astonishing conclusion was reached that these two distinct substances have the same ultimate chemical composition: they possess the same elements combined together in the same proportions, and yet they exhibit widely different properties. This revolutionary idea was unpalatable to the somewhat rigid Berzelius, until four years later, in 1828, Wöhler showed that a similar relationship exists between ammonium cyanate and urea, and that the first of these substances can actually be transformed into the second. Convinced at last, Berzelius in 1830 coined the word *isomerism* to denote the new phenomenon. Wöhler's work on urea had an even greater significance, for he had now prepared artificially for the first time one of the most typical products of animal metabolism, thus striking the first blow at the prevailing theory of the operation of an imagined vital force in the production of organic substances. And so the Industrial Revolution saw the birth of organic chemistry.

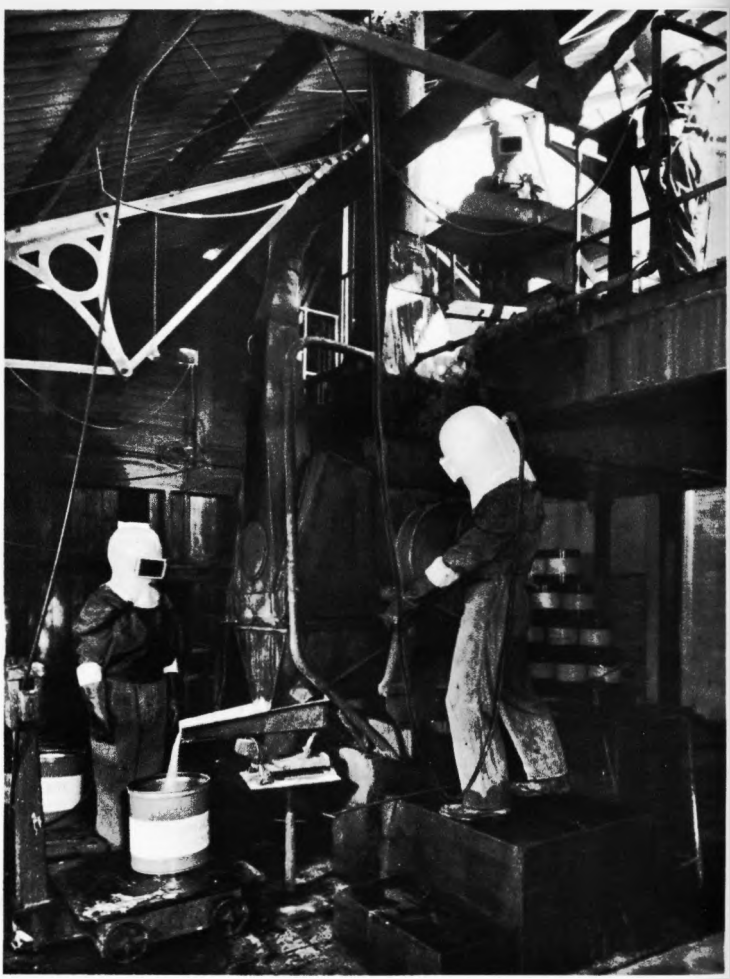
The second half of the nineteenth century was marked by spectacular advances in coal-tar chemistry. This branch of organic chemistry rests upon the theory of molecular structure propounded by Kekulé and Couper in 1858. The arrangement of

1. Screen of Crookes' glass to protect the eyes against electric welders' 'eye-flash (p. 82).

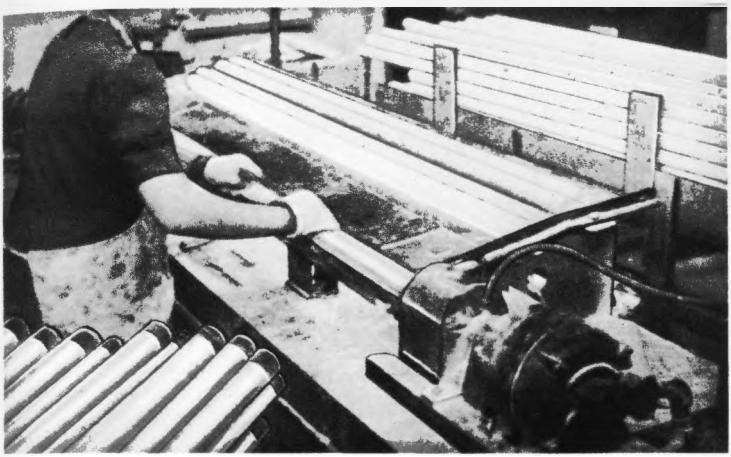
2. Undamaged steel toe-cap of safety-shoe after 350-pound crate fell on to a workman's foot (p. 90).



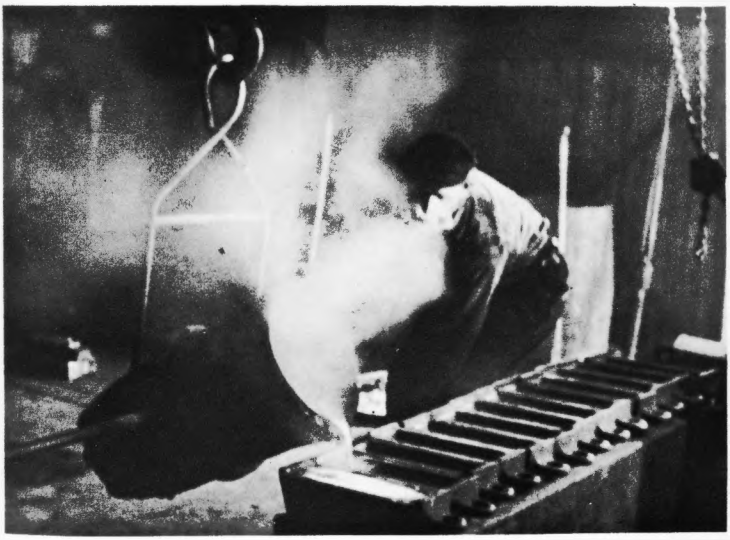
3. Packing plant for dry white lead (p.116) On the left is a completely enclosed grinding mill electrically illuminated from within. On the right are steel packing chambers used for heading up casks. Clean air flows past the operator into the hood above the cask. The hood is connected to the system for exhaust ventilation. Note that the floor is kept wet.



4. Sodium arsenite plant showing protective clothing and air-line helmets worn by the workmen (p. 129).



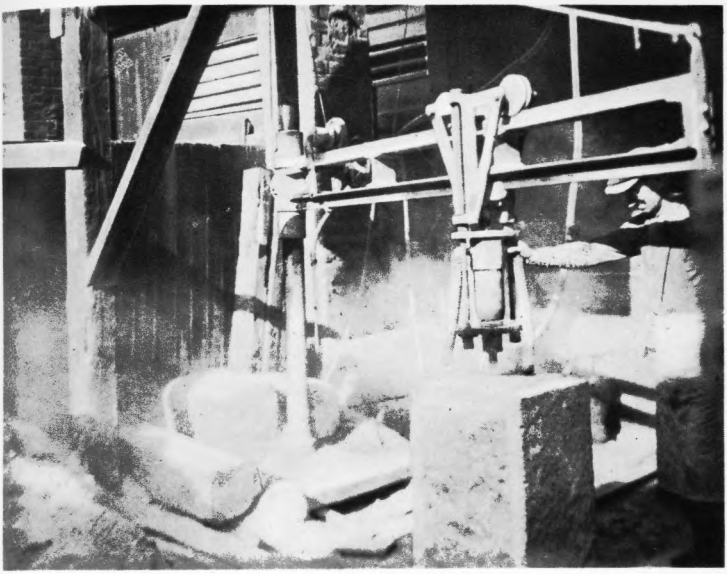
5. Manufacture of fluorescent lamp tubes (p.139). The woman is removing from the end of each tube a white phosphor containing beryllium salts. Exhaust ventilation has been applied locally to the brush used.



6. Fume of cadmium oxide evolved when pouring into moulds a master alloy of equal parts of copper and cadmium. As soon as the danger of chronic cadmium poisoning was realized the fume was removed by exhaust ventilation (p. 142).

7. Acute necrosis of the liver due to poisoning by T.N.T. (p. 153). The great reduction in size can be seen by comparison with the normal liver below. The patient was a boy of 14 who in 1915 filled bags with T.N.T. for 5 weeks. He became jaundiced and died two months after the onset of symptoms.

8. Driver in a gas-proof, air-conditioned tractor cab spraying a wheat-field with D.N.O.C. to kill poppy and charlock (p. 156).

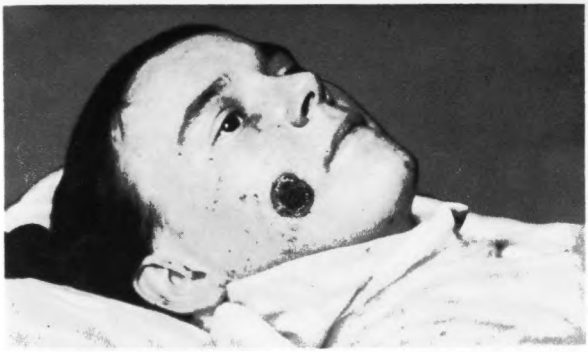


9. Dunter used for smoothing granite (p. 184). Note extensive dust cloud inadequately removed by localized exhaust ventilation through four vertical tubes.

10. Sandblasting cabinet, open only for purpose of photography (p. 199). Note air-line to helmet. The method has been superseded by shotblasting.

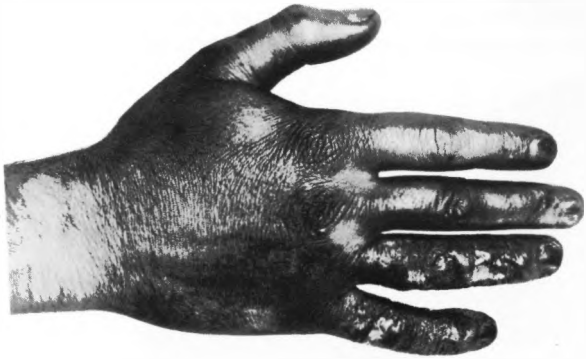
11. Overhead-stoping with a compressed-air drill operated by two miners (p. 205). The drill is provided with an axial water feed which greatly reduces the risk of silicosis.

12. Stripping an asbestos-carding machine before a modern type of exhaust ventilation was applied locally (p. 215).



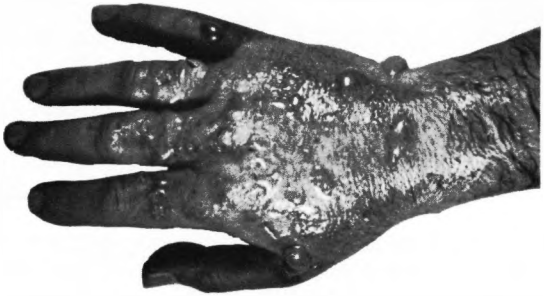
13. Anthrax in a horsehair sorter, showing the so-called malignant pustule on the face on the thirteenth day (p. 220).

14. Cancer on the back of the hand in a man who had begun work 27 years previously in the manufacture of X-ray apparatus (p. 239).



15. Nitric-acid burn and argyria in an unskilled workman employed in dissolving silver in nitric acid (p. 259). He wore a rubber glove which was pierced over the base of the ring finger.

16. Remote handling of radioactive isotopes from behind a lead brick wall looking through a lead glass window (p. 246).



17. Blisters on the skin of the hand of a research chemist twenty-four hours after exposure to phenyl dichlorarsine, an agent intended for chemical warfare (p. 169).

18. Chrome ulcer on the finger of a man who had handled sodium bichromate for 7 years as a colour worker (p. 136).

six carbon atoms as the closed ring of the benzene nucleus was determined by Kekulé in 1866, and substances showing this molecular structure became known as the aromatic carbon compounds. But it was the discovery in 1856 by William Henry Perkin, a London boy of 18, of *the first aniline dye* which prompted England and Germany to develop the distillation of coal-tar on a large scale. The aromatic hydrocarbons and phenols so obtained form the basis of a wide range of synthetic chemical substances, which make up a large branch of chemical industry today.

Synthetic dyes, *drugs*, *perfumes*, and *explosives* are largely manufactured from the products of the distillation of coal-tar, the principal raw materials being benzene, naphthalene, toluene, and anthracene. From these relatively simple organic compounds, a wide range of complex *intermediates* is manufactured. These are made in a number of stages; the first and simpler compounds, such as aniline, nitrobenzene, and nitrotoluene, are known as *primaries* and are made in very large quantities. With the exception of benzene which has deadly long-term effects, most of these substances can be handled without injury to the health of the workmen. Precautions must be taken in the case of the nitro- and amino-compounds, but these are simple and effective.

The extensive use of the aliphatic or open-chain hydrocarbons in modern chemical industry was based on the development of *nitroglycerine*, *dynamite*, and *cordite*. For civil engineering, especially building tunnels, and for blasting rock and soil to provide raw materials such as coal and limestone for industry, such explosives are in constant demand. In 1938 Great Britain used 21,000 tons of high explosives for blasting, a great part of it in coal mines where 63 million shots were fired in the course of one year. Today the production of *nitrocellulose* and *cellulose acetate* is even more important for the manufacture of *lacquers* and *plastics* than for explosives.

The rapid growth of the cellulose-lacquer and moulded-plastics industries has led to the extensive use of many new solvents, most of which were little more than chemical curiosities before about 1925. Amongst these are the *chlorinated hydrocarbons* which have flooded the market, largely because the alkali industry requires an outlet for its by-product chlorine. The various

halogenated hydrocarbons are useful as *refrigerants*, as *degreasers of metals*, *fire-extinguishers*, *cleansers of textiles*, *solvents for rubber*, and *thinners of cellulose lacquers*. Because they are non-inflammable, non-combustible, and non-explosive, they are often labelled *safe*, but although they are safe from causing fire, some of them are far from harmless in their effects on the human body.

The newest development of all is the petroleum chemical industry which has given us new supplies of *olefine hydrocarbons*, *solvents*, *plastics*, *soap substitutes*, and *insecticides*. The use of petroleum as a raw material resembles that of coal. As might be expected from the existence of immense deposits of petroleum within its own frontiers, the United States of America has taken a leading part in this new industry and already uses two million tons annually for this purpose. In Great Britain, although the petroleum chemical industry is of comparatively recent growth, it is already producing many chemical substances originally derived from other sources such as coal and agricultural products. In spite of the stable chemical nature of paraffin hydrocarbons, processes have been developed for their oxidation, chlorination, and nitration, thus producing a wide variety of substances. Whereas the destructive distillation of wood is still carried out to produce methyl alcohol and acetone, and the fermentation of carbohydrates is still used in the production of ethyl, propyl, and butyl alcohols, the petroleum industry now makes all these substances from natural gas and the gases derived from the cracking of petroleum.

Crude petroleum is a mixture of aliphatic, aromatic, and naphthene hydrocarbons, and certain of these are extracted and used directly for chemical production, but the main source of chemical substances is a further type of hydrocarbon, the olefine or unsaturated type, which is produced by the process known as cracking. This process consists of heating a petroleum product to a high temperature and pressure, causing large molecules, such as decane, to break up into smaller ones such as octane, liberating ethylene. Besides ethylene, other gases containing high proportions of olefines, such as propylene, butylene, and amylene, are liberated by cracking. Lately, five cracking plants have been

erected in Great Britain, aimed primarily at ethylene production. As a result the consumption of ethylene has increased tenfold since 1950. It now stands at 70,000 tons annually and by 1960 it may be twice this figure. In the plastics industry, ethylene is polymerized under 1500 atmospheres pressure to make *polythene*. Xylene, necessary for the manufacture of *terylene*, is being made from petroleum.

Soap making is one of the oldest of chemical industries, but today soap has many rivals in the shape of *synthetic detergents*. These are the more significant in a world which is short of fats and alkalis. The very long-chain paraffin hydrocarbons which form paraffin wax can be separated from petroleum by chilling or by solvent precipitation, and then cracked to give olefines. A special variety of the latter process is the production of higher olefines for conversion to synthetic detergents by sulphonation and neutralization, the final products being sodium secondary alkyl sulphates. They are surface active agents or wetting agents with properties like soap, but because they form soluble calcium salts they have the advantage of lathering even in hard water. Since they do not form a scum comparable to the calcium soaps, they are ideal for removing dirt and grease, especially from wool and hair, and as ingredients of shampoos. In the derivation of chemicals from crude petroleum so far few toxic hazards have been encountered.

The twentieth-century physician must know something of the dangers which may occur in the chemical, aircraft, munitions, plastics, and textile industries, and also in agriculture and horticulture. The present rate of industrial development demands the frequent discovery of new materials. The substances produced rapidly become indispensable, but their properties may remain a long time insufficiently understood. Some of them are dangerous under certain conditions; how dangerous has on several occasions not been realized until a fatal accident has occurred. It happens, therefore, that not only doctors practising in industrial areas but also those in agricultural areas may have to deal with patients exposed to substances which until recent years were little more than chemical curiosities, including complex compounds of the aromatic, aliphatic, and olefine hydrocarbons, as well as their

halogenated derivatives, ketones, glycol derivatives, organo-metallic compounds, organic arsenical derivatives, and organic phosphorus compounds.

Some of these are harmless and others so deadly that their use might with advantage be forbidden. Some are absorbed by inhalation, others through the skin; some attack the liver, causing toxic jaundice; others the kidneys, causing suppression of urine; some affect the blood, causing methaemoglobinaemia with lilac cyanosis; others attack the bone-marrow, causing thrombocytopenia, leucopenia, and aplastic anaemia; and still others the nervous system, causing constriction of the visual fields, gross ataxia, astereognosis, and even polyneuritis from inhibition of cholinesterase activity. It follows that the manufacture and use of many organic compounds must be carried out under supervision, strict precautions being used to prevent harm to the operative in factory, warehouse, granary, greenhouse, orchard, and even the open fields.

Poisoning by Coal-tar Derivatives

The coal-tar derivatives are very numerous and complex. The following compounds will be discussed: benzene, nitrobenzene, dinitrobenzene, trinitrotoluene, dinitrophenol, dinitro-*ortho*-cresol, and aniline. It is sometimes possible to predict from the chemical composition of the simpler members of the group what their toxicological action is likely to be. Addition of a nitro- or nitroso-group usually produces a more toxic compound, but it does not follow that toxicity will continue to increase as more nitro-groups are added. Thus, 1-2-4 dinitrophenol is toxic, whereas trinitrophenol is practically harmless. The position of the substituent groups in the benzene ring has a great effect on the toxic action. Thus, the toxic properties of 1-2-4 dinitrophenol are not shared by any of the other isomers. When a nitro-compound is reduced to an amine, as when nitrobenzene is reduced to aniline or nitrotoluene to a toluidine, the toxic characteristics remain much the same, but the intensity of the action is lessened. Sulphonation renders a compound non-toxic; as soon as aniline is sulphonated it ceases to give trouble. The entrance of chlorine

into an aromatic compound does not usually increase the toxicity, as it does in the case of an aliphatic compound. In fact, chlorbenzene is less toxic than benzene.

Benzene

Coal-tar benzene or benzole is obtained as a by-product from the distillation of tar in the manufacture of coal gas. It is the most interesting of the industrial solvents, and with the exception of tetrachlorethane the most dangerous. It must not be confused with benzine, which is unfortunately pronounced in the same way. Benzine is derived from the distillation of petroleum and is non-toxic. Coal-tar benzene has two fields of application in industry: (1) where it is handled in large quantities in closed mechanical systems, as in the distillation of coal-tar, the blending of motor fuel, and in chemical industry; and (2) where it is used as a solvent, as in the rubber industry, artificial-leather manufacture, the dyeing and cleaning industry, the manufacture of paints, varnishes, celluloid, artificial manure, and glue.

Acute benzene poisoning usually occurs as the result of the breaking of distilling apparatus, or in the cleaning of tanks. In slight cases there is giddiness, and a stage of excitement; in severe cases these symptoms may be followed by convulsions, coma, and death. Benzene sinks into the metal of tanks. It is therefore necessary to forbid men to enter such tanks until they have been washed clean and exposed to the open air for many days. Even then a workman lowered into such a tank must be equipped with body belt, life-line, and breathing apparatus communicating with a hose to the exterior. In the stage of excitement the victim may shriek, sing madly, and fight with the rescuer, who, on account of his greater exertions, runs more risk than the rescued.

Chronic benzene poisoning has always been very rare in Great Britain. Speaking for the whole world, however, it was at one time the most important industrial poison after lead. Benzene poisoning may begin with giddiness, nausea, anorexia, weakness, and nervousness. These symptoms are followed by progressive anaemia, with haemorrhagic manifestations, such as bleeding gums, epistaxis, menorrhagia, purpura, and visceral and retinal

haemorrhages. Ulcers or gangrenous patches on the lips, fauces, and pharynx are common. Although poisoning may occur only after many years' exposure there have been fatalities after a few weeks' exposure. The victims of industrial poisoning often constitute a small minority of the workers; a single susceptible individual may contract fatal poisoning in an environment which does not give rise to even mild poisoning in others. The factors responsible for the great variations in susceptibility are largely unknown, but in general, women, especially young women, are more susceptible than men. Changes in the blood may begin from 2 days to 1 month after the first exposure, according to the amount absorbed, and they may progress, or even develop after exposure has ceased. Acclimatization to the vapour of benzene does not seem to occur, and it is doubtful whether any concentration greater than zero is safe over a long period of time.

The bone marrow is attacked, and it may be aplastic, normal, hyperplastic, or leukaemic, the corresponding changes being reflected in the peripheral blood. In the early stages of poisoning there may be little or no evidence of damage to the red cells, but only purpura with leukopenia and granulopenia. The total leucocyte count may fall to a very low level; in fact, the white cells may almost disappear. A count of 1000 per c.mm. is common. In some cases the granular leucocytes fall as low as 10 per cent. The bleeding time may be increased to half an hour, with a corresponding drop in the platelets. The red cell count may fall as low as three-quarters of a million per c.mm. Splenomegaly may appear in protracted cases of poisoning. Myeloid leukaemia confirmed by post-mortem examination has been found in several cases. Necropsy usually shows aplasia of the bone marrow. Haemorrhages may be found in the skin, pericardium, pleura, alimentary tract, meninges, bladder, and uterus. Gangrenous stomatitis and even necrosis of the gastric mucosa have been recorded. Benzene cannot be discovered in the body after death.

Benzene is so toxic that its substitution wherever possible by other solvents which are harmless is, in *preventive treatment*, the method of first choice. In all processes involving the presence of benzene the value of frequent periodical medical examination, including examination of the blood, has been proved by experi-

ence. The more complete and effective the prevention of the escape of vapour into the workrooms, and the more efficient the daily supervision of the ventilating apparatus, the less is the necessity for periodical medical examination.

In acute benzene poisoning the usual methods of resuscitation are used: rest, warmth, artificial respiration, administration of oxygen, and injection of nikethamide as a respiratory stimulant. The patient should be prevented from returning to work too soon.

Cases of chronic benzene poisoning must be treated by repeated blood transfusions, and it must be remembered that the toxic influence may persist even after removal from exposure. The results of treatment are so poor as to convince all who have studied the subject that the use of benzene in industry must be ruthlessly suppressed, except where the process used is entirely enclosed. Fortunately in Great Britain it has been possible to do this.

Nitrobenzene

Nitrobenzene (oil of mirbane; artificial oil of bitter almonds) is used in the manufacture of aniline, in perfumery, and as a flavouring agent, being the chief substitute for oil of bitter almonds. In factories death has taken place from absorption through the skin, where the oil has been spilled on the clothing. The victim of such an accident should be stripped promptly of his clothes, sponged with weak acetic acid or vinegar, and provided with a shower bath and clean clothes. The *symptoms* vary according to the acuteness of the poisoning. Pallor appears, quickly followed by lividity. Unconsciousness occurs with great rapidity, perhaps in 20 minutes. The patient is generally deeply cyanosed, and the blood is dark and viscid, and will sooner or later show the spectrum of methaemoglobin. Death occurs in coma, or recovery takes place after a variable period of unconsciousness. However, nausea and vomiting may begin again after a few days, and toxic jaundice appear. The blood picture is that of anaemia with granular degeneration of the red cells (stippling), and sometimes the presence of nucleated red cells. In severe anaemia a blood transfusion may be considered.

Dinitrobenzene

Dinitrobenzene is used in the manufacture of dyes, and is itself an important explosive. It is a solid, and in consequence poisoning develops less rapidly and is less severe than in the case of nitrobenzene. Poisoning occurs amongst men who either shovel or melt dinitrobenzene. In a mild case there is a sense of pressure in the head, which increases to a violent throbbing headache, giddiness, and dyspnoea. In severe cases the face is deeply cyanosed; the lips, tongue, and ears are deeply purple; and there are nausea, sometimes vomiting, abdominal pain, a staggering gait, and extreme weakness. An attack rarely occurs during work; the man is usually overcome some hours after he has left the plant. Cyanosis occurs, and is accompanied by anaemia with marked stippling of the red cells. Methaemoglobin, porphyrins, haemoglobin, and even albumin have been found in the urine. The smoky colour of the urine may be noticed by the men themselves soon after their first contact with nitrobenzene or aniline. Only very rarely does toxic jaundice occur. It is well known in industry that cases of poisoning are more frequent in hot weather than during the colder seasons of the year. Lack of care and unclean habits are predisposing causes. Those who do not change their working clothes on returning home may sit before the fire and absorb the poison from the evaporation of crystals or from the material in solution on their clothing. Absorption from the alimentary canal is more rapid if the stomach is empty, and it is therefore desirable that men should take a meal before they work. Alcohol undoubtedly favours absorption, and several instances are on record in which poisoning has supervened after indulgence in ordinary amounts.

Trinitrotoluene

Trinitrotoluene is handled mainly in the filling of shells. The first *symptoms* of poisoning are drowsiness, headache, nausea, loss of appetite, epigastric pain, vomiting, and giddiness. There is some cyanosis of the lips, followed by dyspnoea with marked drowsiness and staggering gait. Dermatitis in the form of a diffuse erythema sometimes occurs on the dorsal surfaces of the wrists,

and on the face and neck. The symptoms come on gradually after several days' or weeks' work, or they appear on a hot day after a few hours' work and cause collapse. If cyanosis occurs in 1 in 10 of the workers, toxic jaundice probably affects 1 in 500. The greatest incidence of jaundice is in the third month of employment. Premonitory symptoms, such as drowsiness, giddiness, depression, and dark urine, are sometimes present, but the onset is often quite sudden. Sometimes a latent interval occurs between removal from exposure and the onset of jaundice. The *prognosis* is always uncertain, but grave symptoms of hepatic insufficiency sometimes appear rapidly. The mortality is 25 per cent. The morbid appearances are those of yellow and red necrosis of the liver, with great reduction in its size and weight (Plate 7). The necrosis of the liver cells is associated with infiltration and subsequent fibrosis resembling ordinary portal cirrhosis. Aplastic anaemia sometimes occurs among trinitrotoluene workers, but its incidence is very small. The latency of the blood changes is even longer than the latency of the jaundice, for it is found that anaemia can develop as long as 9 months after exposure to trinitrotoluene has ceased. The anaemia is usually, if not always, fatal. At necropsy fatty marrow is found throughout all bones. There is an excess of iron pigment in the liver, and multiple haemorrhages are found in the tissues. The skin is the main channel of absorption. Experience in industry goes to show that when a poison is absorbed by this route the application of *preventive measures* is most difficult. The principles involved are cleanliness of the air breathed, secured by effective ventilation or filtration through an effective respirator; cleanliness of the implements used and cleanliness of the person secured by protective clothing and by personal attention to the skin. Filling factories must have their own laundries and each worker must have two lockers, one for his own clothing and the other for his protective clothing. Workers and supervisors must be specially trained for the job. Periodical medical examination is essential.

Dinitrophenol

Of all the dinitrophenols it is only the 2-4 or *alpha* isomer which has toxic properties. It is a pale-yellow crystalline powder,

which is used as an explosive, in the dye industry, and for the preservation of timber. Absorption takes place through the respiratory tract, the alimentary canal, and the skin. Heat aids absorption and alcohol increases the incidence of toxic manifestations. D.N.P. acts by stimulating the general metabolism. Workers show yellow staining of the face, legs, and forearms, and especially of the palms and soles. Staining of those parts of the body which have not been in direct contact with the powder indicates a dangerous accumulation of the compound in the body. In a small proportion of workers a pink maculo-papular eruption appears on the exposed skin. Mild poisoning is characterized by lassitude, slight headache, night sweats, and fatigue on the slightest exertion. Workmen may lose weight from the time that they first take up the work. Acute intoxication comes on suddenly, with a sensation of extreme weakness in the limbs and painful constriction in the chest, a burning thirst, abundant sweats, and an agitation and anxiety which are characteristic. Other signs are pallor, dyspnoea, and scanty urine, which may be a deep orange colour owing to the presence of 2-amino-4-nitrophenol. In more severe cases death may take place within a few hours after a rise of temperature to 104° F. or over. The victim has severe sweating, intense thirst, and sometimes colic and diarrhoea. The basal rate of metabolism is increased to 200 per cent or more. The state of anxious terror and restlessness is followed by hyperpnoea, coma, convulsions, and death. Necropsy reveals no characteristic lesion. When the dose is not fatal the symptoms rapidly decrease and many workers develop a tolerance to the poison. The workmen must be provided with a complete set of underclothing and overalls into which they change from their working clothes, a separate cubicle being provided for each man. Well-designed exhaust ventilation must be applied locally to take away the fumes in the melting of the compound and also in the filling of shells. Any dust that collects around the margin of the shell must be removed by a vacuum cleaner.

In 1933 dinitrophenol became widely used in the treatment of obesity, especially in the U.S.A. A dose of 3 mg. per kg. of bodyweight will cause a rise in basal metabolic rate and loss of weight, unattended by tachycardia. Toxic symptoms were soon reported,

including urticaria, exfoliative dermatitis, jaundice, peripheral neuritis, loss of power to discriminate between sweet and salt tastes, fullness in the ears, deafness, fall in blood pressure, albuminuria, neutropenia, and fatal agranulocytosis. After dinitrophenol had been employed for 4 years, cataract was found to be a late complication of the use of the drug. It appears from 3 to 18 months after the first dose is taken, the change is bilateral, and the lens fibres alter so quickly that the cataract swiftly progresses to total blindness. This final disastrous effect of dinitrophenol brought to a close the unfortunate popularity of this drug.

Dinitro-ortho-*Cresol*

The compound 4:6-dinitro-*ortho*-cresol is a yellow solid, manufactured on a large scale as a weed-killer, insecticide, ovicide, and fungicide. It is applied in agriculture as an aqueous solution of the sodium salt, while for locust control it is used as a dust or solution in oil. Its use in the treatment of obesity, although effective, has been given up because it is too dangerous.

D.N.O.C. may be considered to be about twice as toxic as dinitrophenol (D.N.P.). It is a cumulative poison in man and is eliminated slowly. Like D.N.P., its lethal effect is due to excessive stimulation of the general metabolism. At necropsy the changes noted are yellow pigmentation of all the tissues, dehydration, petechial haemorrhages of the brain and lungs, and parenchymatous degeneration of liver and kidneys. A papular dermatitis is common in workers handling D.N.O.C., and nasal irritation and burns of the skin of the hands have been reported. The *earliest symptom* of poisoning is an exaggerated feeling of well-being, but this is difficult to assess. It is likely to be present when the concentration of D.N.O.C. in the blood is of the order of 20 micrograms per gram of blood. In severe cases unusual thirst, excessive sweating, and fatigue are followed by weakness, high fever, tachycardia, anxiety, and great hyperpnoea. The basal rate of metabolism may rise even as high as 400 per cent. There is loss of weight, perhaps 20 pounds in a few weeks. Severe liver damage has sometimes occurred, but cataract has been recorded once only.

Measures adopted to reduce the incidence of D.N.O.C. poisoning include: (1) periodical medical examination of the workers,

(2) the introduction of locally applied exhaust ventilation in factories, (3) the use of masks, protective clothing, and enclosed tractor-cabins by spray operators (Plate 8), and (4) the exclusion from further contact, for at least 6 weeks, of workers in whom the concentration of D.N.O.C. in the blood is 20 micrograms or more per gram. No antidote to D.N.O.C. is known. Early diagnosis is essential, and *treatment* on general lines will result in recovery, even in seriously poisoned patients. The patient must be kept cool by tepid sponging; fluids and electrolytes, which are lost in the profuse sweating that characterizes the illness in its acute stages, must be replaced; barbiturates should be administered in doses adequate to allay the anxiety.

Aniline

Aniline is a colourless, oily liquid which turns dark brown on exposure to light or air. It is handled in the manufacture of dyes, in the dyeing and cloth-pressing industries, in the extraction of resin, and in the rubber industry. Aniline poisoning arises usually from inhalation, but absorption through the skin, and, less frequently, inhalation of dusts of aniline compounds may cause it. Care must be taken to change the clothes at once whenever they are splashed with aniline. Men must avoid entering chambers filled with its vapour. The *symptoms* of aniline poisoning are similar in all respects to those of nitrobenzene poisoning. The convenient term *anilism* may be used to cover the symptoms produced by most of the nitro- and amino-derivatives of benzene.

In *acute aniline poisoning* there is headache, weakness, difficulty in breathing, cyanosis, loss of power in the limbs, and giddiness. In severe cases the cyanosis is more intense and prostration occurs with a cold moist skin, small pulse, air hunger, and even death in coma. When recovery occurs it is often gradual and may be accompanied by increased frequency of micturition. In *chronic poisoning* the workers show slight cyanosis, secondary anaemia, and sometimes sleeplessness, headache, giddiness, and abdominal discomfort. In hot weather practically all the men exposed to aniline and similar compounds in a dye works show slight cyanosis owing to methaemoglobinaemia.

The manufacture of nitrobenzene, and the reduction of nitrobenzene and nitrotoluene to aniline and toluidine must take place in closed vessels. Even so, escape of small quantities of aniline into the atmosphere is very difficult to prevent unless ample ventilation is provided. Therefore, in addition to the technical regulations, there must be insistence on cleanliness of the workrooms, personal cleanliness on the part of the workers, and provision of baths and changes of clothing. Contact with aniline and nitrobenzene, especially on the skin, and also the spilling and splashing of these fluids must be carefully avoided. All workers must be instructed as to the symptoms of nitrobenzene and aniline poisoning, and the right steps to take if poisoned. Regular medical inspection of workmen is desirable.

Workers, and especially those newly employed, must be under supervision in order that assistance may be rendered them on the first signs of poisoning. Medical assistance should be within easy reach. Systematic instruction should be given in first-aid methods, and in the use of apparatus for oxygen and carbon dioxide inhalation. The possibility of skin absorption must always be borne in mind. A victim whose skin or clothing has been splashed with aniline may turn blue in the face and begin to stagger. Someone may take him out to the fresh air or administer oxygen, when what he needs most is to have his clothes stripped off and be given a bath. Workers entering stills and similar chambers should always be equipped with breathing apparatus and a supply of oxygen. Other aids, such as safety belts which are held by helpers, involve certain risks, especially as the rescuer is easily induced to spring to the assistance of his unfortunate mate without the necessary breathing equipment. The frequency of such accidents calls urgently for the use of breathing apparatus.

Poisoning by Halogenated Hydrocarbons

Numbers of halogenated hydrocarbons are already in use and new ones are constantly being introduced. The following compounds will be discussed: methyl chloride, methyl bromide, carbon tetrachloride, tetrachlorethane, trichlorethylene, and the chlorinated naphthalenes. They vary greatly in their toxic effects,

trichlorethylene being relatively harmless and tetrachlorethane deadly. The action of carbon tetrachloride has been extensively studied because of its use in the treatment of hookworm disease. Tetrachlorethane dramatically attracted attention in the aircraft industry in England in 1914. Methyl bromide caused fatalities in the Swiss chemical industry in 1920. A mass poisoning from leaking refrigerators in Chicago in 1929 led to an increase in knowledge of the action of methyl chloride. Trichlorethylene has been studied because of its extensive use in dry cleaning. It led to trouble in German industry in 1931. Chlorinated naphthalenes caused no serious harm until 1937.

Methyl Chloride

Men working in chemical plants or employed upon making, installing, or repairing refrigerators may be exposed to methyl chloride. *Symptoms* of poisoning include giddiness, weakness of extremities, nausea, vomiting, restlessness, followed by somnolence and then by dimness of vision, which may not clear up until 14 days after removal from exposure. Later there is some rise of temperature, pulse, and respiratory rate, usually with oliguria and occasionally with suppression lasting up to 48 hours. Evidence of acute nephritis is found in about half the cases. Anaemia may be found, the red cells dropping as low as 3 million per c.mm., and the haemoglobin as low as 50 per cent. The death-rate is as high as 35 per cent.

Methyl Bromide

Methyl bromide is a colourless, non-inflammable gas, but it comes on the market compressed into containers, in which it assumes the liquid condition. It is valuable as a methylating agent, a fire extinguisher, refrigerant, fumigant, and insecticide. Methyl bromide is a deadly and insidious poison with a delayed action like phosgene and nitrogen dioxide. The latent interval varies from 4 to 48 hours, and then the *symptoms* begin abruptly with nausea, vomiting, headache, vertigo, dimness of vision, diplopia, euphoria, and delirium. In severe cases pulmonary oedema, oliguria, suppression of urine, convulsions, and even acute mania may occur. The skin is pale, the temperature normal, sweating is

profuse, trismus and even opisthotonus may be seen, and the pupils are dilated. The skin may be affected in susceptible people; premonitory pruritus is characteristic and may be intense. Methyl bromide burns are superficial, rarely extending deeply enough to destroy the whole dermis. They are characterized by excessive vesication with reddening and swelling of the surrounding skin. Healing readily occurs, and in most cases there is considerable desquamation. Mild cases usually recover, but where there is oedema of the lungs, convulsions, anuria, or severe burns the outcome is often fatal. In cases showing mild systemic symptoms *treatment* by rest in bed for 2 days with 3 weeks' convalescence will usually be adequate. If there is cyanosis little can be done except to administer oxygen. The skin lesions should be treated with 2 per cent tannic acid in triple-dye solution or with propamidine isethionate cream.

Carbon Tetrachloride

Carbon tetrachloride is used in industry as a solvent for fats and rubber, for dry cleaning, for cleaning oil from machinery, and under the name Pyrene as a fire extinguisher. Acute and sometimes fatal poisoning has occurred from the anaesthetic effects of carbon tetrachloride used as a dry shampoo for the hair. Men exposed to the vapour sprayed from Pyrene fire extinguishers in confined spaces have suffered from oliguria and jaundice. In animal experiments carbon tetrachloride has been shown to cause necrosis of the liver. In man it may give rise to acute nephritis, necrosis of the liver, oedema of the lungs, or retrobulbar neuritis. The early stages of the illness are characterized by persistent headache, nausea, vomiting, diarrhoea, and tenderness over the liver. Such *symptoms* are often followed by oliguria, suppression of urine, and uraemia. Sometimes the clinical picture closely simulates that of the acute abdomen. The blood urea may rise to 300 mg. per 100 ml., and the patient, though practically moribund, may suddenly develop polyuria and recover even after almost complete anuria lasting 10 days. When the liver is attacked recovery may follow an attack of jaundice lasting as long as 2 months. In the *treatment* of cases rendered unconscious by acute poisoning, it is important that the patient should not be placed

upon the floor of the room where the accident occurred, for the vapour is five times denser than air and therefore accumulates on the floor. Hepatic insufficiency should be treated by glucose drinks, together with large doses of calcium lactate up to 15 g. a day. Calcium gluconate may be given by intramuscular injection. Protein hydrolysates, methionine, and vitamin supplements have also been used.

Tetrachlorethane

Tetrachlorethane is the most dangerous of all the chlorinated hydrocarbons, being nine times as toxic as carbon tetrachloride. It is a very good solvent for cellulose acetate, which, being non-inflammable, is used for purposes for which cellulose nitrate (celluloid) is not adapted. Cellulose acetate was the chief constituent of the dope used as a waterproof coating for the wings of aeroplanes in the First World War, and it is now used to make non-inflammable cinema film. The *symptoms* of poisoning are general malaise, loss of appetite, nausea, headache, and constipation. After several days or even weeks, jaundice develops and vomiting is then likely to become more marked. In fatal cases necrosis of the liver is found in the form of acute red and yellow atrophy. In one case the liver weighed only 19 oz. The blood changes in mild poisoning consist of an increase of large mononuclear cells up to 40 per cent, with a slight elevation of the white count. Blood counts have been taken in order to detect early poisoning. Elaborate exhaust ventilation in factories and workshops fails to prevent toxic jaundice. It is therefore necessary to use a ***harmless substitute*** such as amyl acetate.

Trichlorethylene

Trichlorethylene is employed extensively in dry cleaning and as a degreasing agent. It has assumed an important place in the list of fat and rubber solvents, displacing to some extent carbon tetrachloride. Trichlorethylene has a powerful narcotic effect. The workman affected is usually found unconscious on the floor, and if there has been prolonged exposure to a large dose the effects may be fatal. Chronic exposure has been held responsible for paralysis of the sensory fibres of the fifth nerve, and also for

retrobulbar neuritis followed by optic atrophy. An indirect form of injury to the eye sometimes occurs when a foreign body lodges in the cornea and produces ulceration. The workman is not aware of the presence of the irritant substance, because the cornea has been rendered insensitive. Trichlorethylene is not so likely to attack the liver as tetrachlorethane. Toxic jaundice and albuminuria have been recorded only rarely. Dry-cleaning establishments employing trichlorethylene should be provided with efficient exhaust ventilation. When solutions containing trichlorethylene are applied to the interior of closed vats the men should work in pairs, relieving each other frequently. The man in the enclosed space should be provided with a lifebelt and also with an apparatus through which he can breathe air from outside.

Chlorinated Naphthalenes

When naphthalene is chlorinated a series of wax-like substances is produced. These are used as an insulating coat on wires or on metal bars to circumscribe the action of plating processes. They may produce acne, starting on the face and around the angles of the jaws and malar prominences, and spreading on to the sides of the face, neck, shoulders, and forearms. The skin lesions in a typical case are comedones, papules, pustules, and, in severe cases, small cysts. Since 1936 several cases of jaundice have occurred in workers handling chlorinated naphthalenes. In one fatal case necropsy showed acute red and yellow necrosis of the liver, which weighed 650 g., the normal being 1500 g. By attention to ventilation, protective clothing, and medical supervision of workers, the chlorinated naphthalenes can be handled in industry with safety.

Poisoning by Tetra-ethyl Lead

Tetra-ethyl lead is an organic, lipoid-soluble compound readily absorbed through the skin and respiratory tract. It is a clear, heavy oily liquid with a peculiar sweetish odour, and is somewhat volatile at ordinary temperatures. It is added to petrol in proportions up to 1 in 1260 as an antidetonant. Twelve per cent of the annual consumption of refined lead is used in its manufacture.

In 1923, when it was first manufactured in the United States of America, 149 cases of encephalopathy occurred in men employed on three separate plants. Within 17 months 11 deaths were reported. Much excitement and alarm were caused, and this led at first to the prohibition of the manufacture. In the Second World War a new hazard arose in the process of cleaning storage tanks which had held ethyl petrol. In England some of these tanks were underground and were of 4000 tons capacity. Twenty-five cases of poisoning by tetra-ethyl lead occurred, two of them fatal. War conditions in countries of the Middle East and Far East made the cleaning of tanks difficult to supervise, and there were 200 cases of poisoning with 40 deaths.

The early *symptoms* include insomnia, loss of weight, anorexia, and morning nausea, but there is no colic. Mental manifestations dominate the clinical picture, and in severe cases restlessness, bad dreams, hallucinations, and delusions are common. Several symptom-complexes have been distinguished – the delirious, manic, confused, and schizophrenic. Weakness, tremor, muscular pains, and ease of fatigue are frequent complaints. The tremors affect the extremities, lips, and tongue, and are coarse and jerky, and aggravated both by effort and by attempts at control. With severe exposure there may be the abrupt onset of acute maniacal symptoms with suicidal tendencies or the occurrence of a convulsion. Less severe cases begin with insomnia, sleep being difficult, broken, and restless, sometimes with wild and terrifying dreams. By day, mental excitement may be marked, headache is usual and often severe, and vertigo is frequent. Blurred vision and diplopia owing to weakness of the extrinsic ocular muscles are occasional complaints. Evidences of meningeal irritation are absent; the cerebrospinal fluid may at times be under increased pressure, but it is not otherwise abnormal. Punctate basophilia is absent or slight. In the patients who recover, all symptoms disappear in from 6 to 10 weeks. Occasionally an anxiety state persists for a time.

The sedative action of repeated doses of barbiturates together with adequate fluid intake are the *essentials in treatment.* Morphine is contra-indicated. Glucose, 5 per cent in saline, may be given intravenously up to 3 litres a day, and if it is given as a

drip, hexobarbitone may be added. In severe cases from 2 to 4 g. of magnesium sulphate in 2 per cent aqueous solution should be given intravenously accompanied by doses of pentobarbitone sodium up to gr. 15 daily by mouth. Enemata of 6 oz. of a saturated solution of magnesium sulphate often have a sedative effect when they can be retained.

As to *preventive measures*, by meticulous attention to detail it is possible to manufacture tetra-ethyl lead and to blend it with petrol without risk to the workers. Both manufacture and blending are carried out in closed systems. Elaborate precautions are taken in transport, storage, and handling of the fluid, and great care is exercised to avoid leakage or spilling. In blending and laboratory work impervious gloves and respirators are used. Strict regulations must be laid down for the cleaning of tanks which have contained leaded petrol. Those responsible should make it quite clear that such work is never to begin without reference to some authorized person. This makes it possible to do the work under supervision and to use trained workmen properly equipped with protective clothing. Although ethyl-petrol contains less than one part in a thousand of tetra-ethyl lead it should not be used for cleansing the skin or for dry cleaning, and to prevent this it is coloured by a dye. While decarbonizing engines which have burned leaded petrols, mechanics must wear dust masks. Routine medical examination of workmen and technicians should be carried out wherever possible.

Poisoning by Organic Mercury Compounds

Organic compounds of mercury were first used in chemical research in 1863, in therapeutics in 1887, and in the manufacture of seed dressings and other fungicides in 1914. Those with hydrocarbon groups of low molecular weight have been found the most toxic, and the only cases of systemic poisoning recorded in man have been due to methyl and ethyl derivatives.

In 1863 di-methyl mercury was used in the course of some research work undertaken at St Bartholomew's Hospital in London to determine the valency of metals and metallic compounds, and 2 laboratory technicians engaged in this work

developed symptoms of poisoning and died. One of them was a German aged 30 years who had been exposed to di-methyl mercury for 3 months. He complained of numbness of the hands, deafness, poor vision, and sore gums. He was found to be slow and dull in manner, unsteady in gait, and unable to stand without support. There was no motor palsy and the fundi were normal. Within a week he became rapidly worse, restless, unable to answer questions, incontinent of urine, and comatose. He died 2 weeks after the onset of symptoms.

A second technician, aged 23 years, had worked in the laboratory for 12 months and had handled di-methyl mercury for a period of 2 weeks only. The record does not state how soon after exposure he became ill. It could have been from 2 days to 2 months. He complained of sore gums, salivation, numbness of the feet, hands, and tongue, deafness, and dimness of vision. He answered questions only very slowly and with indistinct speech. There was ataxia, but no weakness of the upper limbs. Three weeks later he had difficulty in swallowing, was unable to speak, had incontinence of urine and faeces, and was often restless and violent. He remained in a confused state and died of pneumonia 12 months after the onset of symptoms. A third technician was affected with symptoms similar in character but less severe. He eventually recovered. The story of these deaths has been handed down verbally from one generation of chemists to another.

In 1887 animal experiments were carried out which suggested that di-ethyl mercury was highly toxic. The picture it caused of poisoning in animals was found to differ from that of poisoning by inorganic mercury compounds. There was only moderate inflammation of the intestinal tract, but the nervous system was constantly involved. An ascending paralysis was combined in some animals with ataxia. Incoordination of movement was noticed especially in rabbits, and motor paralysis in dogs and cats. Tremor, blindness, loss of the sense of smell, transient deafness, and attacks of wrath on the slightest provocation were also noticed in many of the dogs.

Seed-borne diseases of cereals and of flax are often treated by organic compounds of mercury. They are used in the prevention

of such diseases as bunt of wheat (*Tilletia tritici*), covered smut of barley (*Ustilago hordei*), leaf stripe of oats (*Helminthosporium avenae*), leaf stripe of barley (*Helminthosporium gramineum*), foot rot of flax (*Phoma*), stem break of flax (*Polyspora lini*), seedling blight of flax (*Colletotrichum lini*), and flax wilt (*Fusarium lini*).

The relationship between the molecular structure and the fungicidal activity of organic compounds of mercury has been investigated. In 1923 the minimum concentration of different compounds necessary to inhibit germination of bunt spores under standard conditions was determined. The importance of molecular structure in determining the fungicidal properties of these compounds also was demonstrated. Thus, inhibition of germination under standard conditions was produced by different compounds in the following proportion – mercuric chloride 0·025, chlor-phenol mercury 0·07, and methyl mercury iodide 0·001. Methyl mercury iodide was thus the most active of the compounds tested, but was discarded by plant pathologists on the score of its highly poisonous effect *on the germination of seeds*. In 1935 plant pathologists carried out further experiments employing tolyl, phenyl, ethyl, and methyl mercury compounds against a large number of seed-borne diseases of cereals, which confirmed the view that the fungicidal properties decrease with increase of the molecular weight of the hydrocarbon group.

The manufacture of phenyl and tolyl mercury acetates in large quantities by the chemical industry in Great Britain and in Germany has been carried on by automatic methods in enclosed apparatus. The products are used mainly in the form of dusts, though sometimes they are employed in solution. So far as is known, no mishap worse than an occasional burn on the skin has occurred in handling them. If an organic mercury compound comes in contact with the skin, warmth and redness occur after 6 hours, and blistering after 18 to 24 hours. The blister contains serous fluid, and the lesion remaining after it bursts may take 3 weeks to heal.

In 1940 42 cases were described of dermatitis among lumbermen who were applying a fungicide to newly cut timber in order to combat stain-producing fungi. The substance used was

an aqueous spray containing one part in 6600 of ethyl mercury phosphate. The hands and forearms of all but a small percentage of the men exposed became red and swollen, and then covered with blebs more than an inch across, simulating burns. The disability lasted from 5 to 30 days.

In 1940 4 cases were recorded of poisoning by inhalation of methyl mercury compounds in a factory in London where fungicidal dusts were manufactured without the use of enclosed apparatus. With the exception of tremor, the symptoms of poisoning by metallic mercury – namely, salivation, stomatitis, and erethism – were absent and the nervous system alone was involved. There was severe generalized ataxia, dysarthria, and gross constriction of the visual fields, while memory and intelligence were unaffected. One of these men was still disabled 20 years after exposure had ceased.

Experiments on rats and a monkey confirmed the selective effect of methyl mercury iodide and nitrate on the nervous system. There was an intense and widespread degeneration of certain sensory paths of the nervous system, the peripheral nerves and posterior spinal roots being affected first, the posterior columns and the granular layer of the middle lobe of the cerebellum later. The most severely affected of these 4 patients died 15 years after exposure had ceased. At necropsy the generalized ataxia was found to be due to cerebellar cortical atrophy, selectively involving the granule-cell layer of the neocerebellum, while the concentric constriction of the visual fields was correlated with bilateral cortical atrophy in the area striata.

In 1943 in Canada 2 girls died who were stenographers in a warehouse which stored di-ethyl mercury for use as a fungicide. Their desks were about 15 feet from a stock pile of 20,000 pounds of the fungicide. At a point 3 feet from the di-ethyl mercury and 30 feet from the floor the atmosphere contained 2·7 mg. of mercury per cubic foot of air. Exposure had occurred for 6 months, but no clinical details were given.

In 1948 5 cases of poisoning by methyl mercury compounds occurred in Sweden, including 2 deaths. Of these, one handled methyl mercury iodide in a factory, and he was affected in spite of observing all the protective precautions in detail. Three of the

men packed a seed dressing containing methyl mercury hydroxide and one of these died. The fifth man died after repeatedly impregnating wood by spraying it with a 0·25 per cent solution of methyl mercury hydroxide. In all cases the illness began with tinglings and numbness of the fingers and lips, and when it progressed further the patient developed gross incoordination of movement which ultimately interfered with both gait and speech. One of the men developed concentric scotomata of the fields of vision and later became completely blind.

Death due to poisoning by ethyl mercury phosphate was reported in 1955 in a nursery foreman who had used it to treat *Didymella lycopersici* infestation of tomato plants in greenhouses. In April 1954 he had made up dilute solutions of ethyl mercury phosphate in water and applied the fungicide by hose. The first symptoms occurred in December 1954, and consisted of headache, nausea, and vomiting; ataxia of the lower limbs had developed by May 1955 and he died in July 1955. At necropsy the anatomical and histological changes in the cerebral hemispheres and cerebellum were identical with those found in 1954 in the case of poisoning by methyl mercury compounds described above.

As to *preventive measures* work published in 1940 made it clear that methyl and ethyl mercury compounds are so dangerous that they should *never* be manufactured again. The warning remains unheeded and the grim record of deaths still occurring in many countries is a sad monument to the greed and stupidity of man. Since the phenyl and tolyl compounds of mercury are effective fungicides and since they are less dangerous and can be handled with safety, *only these* should be manufactured. In the factory adequate precautions must be taken to ensure that dusts and vapours of these compounds do not come in contact with the skin and are not inhaled. The use of gloves and respirators is inadequate as a means of protection; the whole process of manufacture, including the final packing of the dust, should be carried out mechanically. Compared to the factory worker the farmer runs little risk. He should be warned that mercurial dressings are poisonous, and should obtain seed which has already been dressed in an enclosed apparatus. But there is considerable

hazard to men spraying crops in fields and greenhouses. For such men the use of protective clothing and other preventive measures is enforced by the provisions of the *Agriculture (Poisonous Substances) (Organo-mercury Compounds) Order, 1956.*

The ataxia and dysarthria of methyl mercury poisoning must be treated by re-educative movements such as teaching the patient to walk on chalked lines. An expert in charge of a speech clinic with patience and the use of a mirror may teach him to speak; and with perseverance in some cases the patient may be taught to use knife, fork, pencil, and even a typewriter.

Poisoning by Organic Arsenic Compounds

Studies directed to chemical warfare, chemotherapy, and the growth of moulds have drawn close attention to the chemistry of organic arsenic compounds. Most of the aliphatic and aromatic arsines employed during the First World War were substances which had been known for some time. The chlorovinyl arsines and phenarsazine chloride have been studied since 1918. In the Second World War the arsenical smokes received a good deal of attention, in particular the aromatic arsenical cyanides. The organic derivatives of arsine can be divided into three groups as follows:

(i) Aliphatic Arsines

The aliphatic chlorarsine derivatives are highly lethal war gases, although in the military sense their aggressive properties are inferior to those of the aromatic arsines. The initial letters following the names of the gases are abbreviations used as military labels.

Methyl dichlorarsine (*MD*) is a colourless liquid with a characteristic odour. Both the liquid and its vapour are lethal substances acting as lung irritants and as vesicants. It is neutralized by chloride of lime and alkalis.

Ethyl dichlorarsine (*ED*) is a colourless liquid becoming slightly yellow on exposure to air and light. It has a characteristic odour which when highly diluted is reminiscent of fruit. It is a lethal

poison acting as a lung irritant and as a vesicant. It is neutralized by chloride of lime.

Chlorovinyl dichlorarsine (*Lewisite*) is a colourless liquid with a faint odour recalling that of geraniums. It is a very powerful war gas having vesicant, lachrymatory, and lung-irritant effects. It is neutralized by water and alkalis.

(ii) Aromatic Arsines

These arsines differ from those of the aliphatic series both in their physical and chemical properties and in their biological action. Although they have a lower toxicity they are more effective as war gases, acting as energetic sternutators and lung irritants even at very low concentrations. Their aggressive action is provoked by finely divided solid particles, which on liberation in the air form highly lethal smokes.

Phenyl dichlorarsine (*MA*) is a colourless viscous liquid with a pungent odour. In air it gradually turns yellow. It has lung-irritant, vesicant, and lachrymatory effects. In chemical warfare it is employed not as a toxic smoke but as a solvent for other gases. Research chemists have suffered accidental blistering of the skin while handling this substance (Plate 17).

Diphenyl chlorarsine (*DA*) is a colourless crystalline solid only slightly soluble in water. It is readily soluble in phosgene and in phenyl dichlorarsine. As a toxic smoke it has lung-irritant, vesicant, and sternutatory effects. In the solid state, in solution, and as vapour it readily produces vesicles on the skin.

Diphenyl cyanoarsine (*DC*) is a colourless, crystalline solid with a slight garlic odour. It has such a low vapour pressure that for purposes of war it must be diffused in the air as a particulate smoke. It is very effective as a lung irritant, a vesicant, and a sternutatory substance.

(iii) Heterocyclic Arsines

The study of the heterocyclic arsines, that is, those containing the atom of arsenic in the nucleus, led to the discovery of substances whose effects as war gases are even more highly lethal than those of the aromatic arsines.

Phenarsazine chloride (*Adamsite or DM*), also known as

diphenylamine chlorarsine, is a yellow, crystalline solid almost insoluble in water. Used as a toxic smoke it is a powerful war gas, having both lung irritant and sternutatory effects.

In Baltimore in 1946 a number of workers exposed to the dust of Adamsite developed intractable dermatitis involving the exposed areas of the face, neck, and arms. An itching, burning, erythematous, papular eruption proved resistant to ordinary forms of treatment. In 6 of the 7 patients the dermatitis had persisted for from 18 to 50 days before admission to hospital.

British anti-Lewisite (BAL) was applied daily as an ointment. It caused intense burning of the affected areas which lasted for less than an hour, after which the patients were greatly relieved from the previous itching and discomfort. After the first day the dose was increased from 100 mg. to 500 mg. The dermatitis cleared completely in from 2 to 8 days with an average of a little over 5 days. In these studies the patients served as their own controls, since the eruption had been present for different lengths of time before treatment and had failed to respond to other forms of therapy prior to the use of BAL.

It is probable that the effect here was due to the systemic action of BAL as well as to its local effect. Later, BAL was applied to unaffected portions of the skin. These inunctions caused no discomfort and therefore comparatively large amounts of ointment could be employed. The results were highly satisfactory and the dermatitis cleared under this method of application as rapidly as when the inunction had been made to the eruption itself. The fact that arsenic excretion in the urine increased during treatment suggested that it had been released from combination with the cells.

In the search for antidotes to combat the arsenical blister gases, full use was made of the progress of knowledge of the intermediary metabolism of carbohydrates in tissue cells. Each step in the breakdown of glucose and glycogen to carbon dioxide and water is controlled by a definite enzyme, and one of the penultimate stages of this degradation is pyruvic acid, a 3-carbon keto-acid. The enzyme system responsible for the oxidation of pyruvic acid is usually called the *pyruvate oxidase system*. This system has

protein and other components, and interruption of tissue metabolism can occur by active interference with the functioning of any of these components by poisons.

It is known that the pyruvate oxidase system contains a component sensitive to very small concentrations of arsenite. It follows that the metabolism of carbohydrate may be poisoned at an important stage by traces of an arsenical substance. This is the predominant way in which arsenic compounds can cause biochemical lesions, and any antidote must be capable of reversing this biochemical damage. On the basis of these facts, it was logical to use the pyruvate oxidase system as well as the more classical method of *in vivo* injection as a test for new antidotes against arsenic, and during the Second World War research upon these lines was initiated and pursued by a team in Oxford led by Sir Rudolph Peters.

Ehrlich had already shown in 1909 that arsenical substances had a strong affinity for sulphydryl groups, and his work led to the observation that when such substances combined with tissue proteins the active sulphydryl group of the latter spontaneously disappeared. The next step was the discovery that when Lewisite reacted with keratin, approximately 75 per cent of the bound arsenic was in combination with 2 thiol groups. Thus it seemed possible that the high toxicity of trivalent arsenic compounds might be due to their ability to combine with essential sulphydryl groups in certain tissue proteins to form stable arsenical rings. Conversely, in a search for antidotes, it was argued that simple dithiol compounds might form relatively stable ring compounds with Lewisite and might consequently compete effectively with the dithiol proteins in the tissues.

As a result of the systematic attack made on this problem, it was found that simple 1:2-dithiols are capable both of exerting a marked antidotal action against the poisoning of the pyruvate oxidase system by trivalent arsenic compounds and, more important still, of reversing this poisoning when once established. Of the numerous dithiol compounds prepared and tested, one in particular, the 2:3-dimercaptopropanol recommended itself as a local decontaminant in combating the arsenical blister gases. This compound, generally known as BAL (British anti-Lewisite),

was compounded in ointment form for use on the skin and eyes, and the value of such preparations was abundantly demonstrated in numerous experimental studies.

Strict *preventive measures* in the manufacture and handling of all these substances must be observed in the factory, laboratory, and practice field. Plant design must be such as to enclose completely the processes of manufacture and to remove toxic vapours by means of adequate exhaust ventilation. Protective clothing is important, and it may include rubber gloves, rubber aprons, and canister gas masks. Solutions of neutralizing agents such as sodium hypochlorite in suitable containers should be ready for immediate application to the skin in case of splashing.

In *treatment* where a research chemist, workman, or soldier at practice is splashed or otherwise exposed to any of these compounds, he should be kept warm and put to bed in uncontaminated clothing. Where the skin has been contaminated, an ointment containing 5 per cent BAL may be applied, using 500 mg. of BAL daily until healing is complete. If application of the ointment causes pain, BAL should be given intramuscularly instead, using 300 mg. of a 10 per cent solution in benzyl benzoate and arachis oil. Subsequently, daily injections of 150 mg. should be given for 2 or 3 days. Should the vapour or smoke cause the lung-irritant effect, the administration of oxygen may be required.

Poisoning by Organic Phosphorus Insecticides

Since 1946, organic compounds of phosphorus have been used as insecticides for the control of insect pests, such as aphis and red spider. Preparations in common use contain tetra-ethyl-pyrophosphate (T.E.P.P.), hexaethyltetraphosphate (H.E.T.P.), diethyl-*para*-nitrophenylthiophosphate (*Parathion*, E. 605 f., D.P.T.F., or *Bladan*), octamethyl pyrophosphoramide (*Schradan* or O.M.P.A.) and *bis*-mono-*iso*propyl aminofluorophosphine oxide (*Mipafox*). Preparations include liquid sprays, dusts, and wettable powders which are diluted before being applied in greenhouses, orchards, and fields.

POISONING BY ORGANIC COMPOUNDS

Insecticides containing organic compounds of phosphorus are poisonous to man and animals. They act as powerful inhibitors of cholinesterase. The insecticidal properties of T.E.P.P., H.E.T.P., and Parathion are similar to those of nicotine. The effects of H.E.T.P. are almost certainly due to contamination of the manufactured product by T.E.P.P. Schradan and Mipafox are systemic insecticides, that is to say they have no direct insecticidal action nor do they inhibit cholinesterase *in vitro*. They are converted in the plant or animal tissues and are effective as insecticides only against pests which eat the plants. In a single dose they are less toxic than nicotine, but the effects of absorbing small amounts of these anti-cholinesterase substances are prolonged and result in increased susceptibility to absorption of further amounts of any cholinesterase inhibitor. All types of preparations penetrate rapidly through the skin, producing only slight irritation at the site of absorption. Exposure to as little as 0·3 g. daily has been estimated as dangerous to man. The lethal dose by mouth for man is approximately 100 mg. of T.E.P.P. or Parathion, and symptoms follow the administration of more than 10 mg. Absorption may also occur from inhalation and ingestion.

The early symptoms of poisoning are mild and non-specific and may include headache, nausea, anorexia, and unusual fatigue. These may be accompanied by pin-point constriction of the pupils. The symptoms are aggravated by smoking or taking food. From 2 to 8 hours later, nausea, abdominal cramps, vomiting, diarrhoea, muscular twitching, coma, convulsions, and signs of pulmonary oedema may develop. Incontinence of urine and faeces is common. Death may result in as short a time as one hour after the onset of symptoms.

About 3 weeks after recovery from the acute phase of the illness, patients may develop paralysis of the limbs similar to that which follows poisoning by tri-*ortho*-cresyl phosphate. Therefore, all patients who have had acute poisoning by organic phosphorus compounds should be kept under close observation until the cholinesterase activity of the blood has returned to normal.

In *preventive treatment* strict precautions must be taken to protect workers engaged in handling these insecticides. Protection is more easily arranged and applied in factories than in field

operations. In factories where an organic phosphorus insecticide is made, mixed with wetting agents, or incorporated in dusts or wettable powders, exhaust ventilation should secure that this substance is absent from the atmosphere. Protective clothing must include overalls, gloves, boots, cap, and underwear, which are laundered each day and changed immediately if accidentally splashed. Ordinary clothing must be protected from possible contamination. Respirators should be available in factories for use in emergency; in field operations they must be worn during dusting operations and the diluting of wettable powders. It is necessary that the workers should wash thoroughly before eating or smoking, and a bath should be taken at the end of a day's work. These instructions, together with an account of the symptoms of poisoning, must appear on the labels of containers of organic phosphorus compounds. The attention of all workers exposed to risk should be directed repeatedly to the toxic properties of these compounds.

Regulations under the *Agriculture (Poisonous Substances) Act, 1952*, and the *Agriculture (Poisonous Substances) Regulations, 1956*, prohibit the use of organic phosphorus compounds unless the men working with them are wearing protective clothing. The number of hours during which workers can be employed are restricted to 10 in any one day and 60 during 7 days. The employment of any worker under the age of 18 is prohibited. The regulations also contain requirements about the provision and maintenance of protective clothing, the provision of washing facilities for workers, the notification of sickness and absence, the training and supervision of workers, and the keeping of registers. Unhappily, no provision is made for the estimation of the blood-cholinesterase activity at frequent intervals of people at risk.

In *treatment* atropine is an antidote to the muscarinic and central-nervous-system effects of this form of poisoning. It should be given in doses of 1 to 2 mg. (gr. $\frac{1}{60}$ to $\frac{1}{30}$) at hourly intervals until the pupils are dilated. Oxygen, under slight pressure to overcome bronchial spasm, should be administered at the first sign of pulmonary oedema. The fibrillary twitching of muscles appears to affect particularly the diaphragm, and artificial respira-

tion may be necessary. Involvement of striated muscle is the result of the nicotine-like action of these compounds. No antidote to this effect is known and death may occur from neuromuscular paralysis, even though the muscarine-like effects and the signs of involvement of the central nervous system have been controlled by atropine.

CHAPTER SEVEN

DUST DISEASES OF THE LUNGS

Silicosis – The Occupations in which Silicosis Occurs – Prevention of Silicosis – Pneumoconiosis of Coal Miners – Asbestosis – Byssinosis

THIS is a group of lung diseases resulting from the inhalation of dust in various trades. They are sometimes called the pneumonokonioses or, more shortly, pneumoconioses. Some of the dusts produce serious lung disease, others are less harmful, and some are benign.

Dusts consist of particles or aggregates of particles suspended in air and measuring from 150 microns to 0·5 microns in diameter. The sand of deserts, the dust of streets, and the pollens of plants are of such large particle size that they become trapped in the nose and upper respiratory passages and never reach the lungs. Industrial dusts result from vigorous mechanical attrition, blasting, grinding, drilling, rubbing, crushing, hammering, and sawing. Particles 5 microns or less in diameter can reach the alveoli of the lungs; in the ashed lung specimens of men who have died of silicosis the most representative particle measures 1 micron in diameter.

Individuals vary greatly in their capacity to deal with dusts, and of two men who have been working at the same job for the same length of time one may get a disease of the lungs and the other may be unaffected. The reasons for the differences in individual reaction to dust are not accurately known, but it is likely that they depend on anatomical, physiological, and biochemical variations from one person to another. It is known, however, that previous damage to the lungs is a factor which leads to the retention of dust in them. In any case, there are instances where people have spent many years in the dusty trades and have died from causes other than the dust diseases. On the other hand, many thousands have died as a direct result of the inhalation of dust.

Of the dusts which do reach the alveoli, silica and fibrous silicates set up different types of fibrous reaction in the lungs and the process is usually progressive. But the dusts of some of the non-fibrous silicatés and of the compounds of certain metals, among them calcium, iron, tin, and barium, seem to be inert when inhaled. They are capable of lying in the lungs for years without inducing any deposition of fibrous tissue or other reaction, but because they are relatively radio-opaque they may produce changes in the x-ray film which must then be distinguished from those of silicosis. The resulting conditions are sometimes referred to as *benign pneumoconioses*. The inhalation of vegetable and animal dusts may cause asthma and bronchitis indistinguishable from these diseases of non-occupational origin. Of the dust diseases of the lungs only silicosis, pneumoconiosis of coal miners, asbestosis, and byssinosis will be considered here.

Silicosis

Silicosis is the most important disease of this group and is defined as a pathological condition of the lungs due to the inhalation of particulate matter containing free or uncombined silica (silicon dioxide). It is important to distinguish between silica in the free state and in the combined state as the various silicates.

Silicosis and pneumoconiosis must not be used as synonymous terms. Logically one could classify the pneumoconioses as pneumoconiosis due to silica, pneumoconiosis due to asbestos, pneumoconiosis of coal miners and so on, but such a method would be too cumbersome. Legal considerations make it important that the term silicosis be reserved for that condition caused by the inhalation of uncombined silica, the special characters of which render it capable of being identified when it occurs in typical form. Dusts other than uncombined silica produce pneumoconioses, but these do not present the characters which are possessed by silicosis in its typical form.

Silicosis is widespread throughout the world. It is prevalent in many industries, and of all the pneumoconioses it claims the largest number of victims, either alone or with tuberculosis with which it is frequently allied. Silicosis has been known as dust

consumption, ganister disease, grinders' asthma, grinders' consumption, grinders' rot, grit consumption, masons' disease, miners' asthma, miners' phthisis, potters' rot, rock tuberculosis, stone-hewers' phthisis, and stonemasons' disease. Stoneworkers' lung is sometimes referred to as chalicosis and slateworkers' lung as schistosis. The origin of these terms is obvious. Recognition of silicosis depends in a given case on a knowledge of the occupational history, on the clinical and the radiological examination, and in fatal cases on the necropsy findings.

Silicosis is generally divided into first, second, and third stages, or slight, moderate, and severe degrees. The first stage, the so-called simple silicosis, supervenes in a workman who has been employed in an industrial process involving exposure to siliceous dusts for a period of many years. The changes can occur from a few months after exposure to over sixty years. Commonly they are found half-way between these extremes. The onset of symptoms is marked by dyspnoea on exertion, slight at first, and later increasing in severity. Throughout the illness dyspnoea remains the most important symptom. Slight cough may be present from the first. It is usually unproductive or with scanty sputum. The general condition of the patient is unimpaired. Physical signs in the chest are slight. Diminished expansion is scarcely, if at all, present. Dullness can rarely be demonstrated, and in older subjects there may be areas of hyper-resonance due to emphysema. There is no alteration of the breath sounds, and there are no added sounds. In this stage, impairment of working capacity may be slight or absent. In the second stage, dyspnoea and cough become established and further physical signs appear. There is diminished expansion of the chest, patchy dullness, sometimes with bronchial breath sounds, and scattered rhonchi, especially at the bases. There is always some degree of impairment of working capacity. In the third stage, dyspnoea leads to total incapacity. Right heart hypertrophy and then failure may supervene.

In the first stage of silicosis the radiograph shows the presence of discrete nodular shadows, circular, and at the most 2 mm in diameter. They may be partially distributed throughout the films, more widespread, or even generalized, but they remain

discrete. Sometimes they are, in part, obscured by emphysema. In the second stage the whole of both lung fields is occupied by nodular shadows, and there is some coalescence to form more or less dense opacities. In the third stage the radiographs indicate areas of massive consolidation. Pulmonary tuberculosis may be present in any stage of silicosis. It may alter the symptoms, physical signs, radiographic appearances, and the whole course of the disease. It is the most frequent accompaniment of silicosis. Since tuberculosis of the lungs may simulate silicosis in radiographs, no diagnosis should ever be made exclusively on radiographic appearances.

At necropsy the lungs are generally large and retain their shape on removal from the thorax. Pleural adhesions are nearly always present, and they may be extensive at the bases. On parts of the lung not covered by thickened pleura, the surface is studded over with pale grey nodules, each of which is felt to protrude above the general surface and to be part of a nodule which extends into the lung substance.

The cut surface of the lung shows excess of pigmentation throughout, but the striking and distinctive feature is the presence of numerous round nodules. These are dense, tough, and black or grey in colour. Each nodule is from 2 to 5 mm. in diameter, but several may be aggregated together to form large composite nodules, or many may be united in a massive fibrosis. In long-standing cases, individual nodules may be thrown into sharp relief by emphysema of the surrounding lung. The centres of the nodules may undergo calcareous change. In cases where exposure to dust has been intense and the course of the disease relatively rapid, the nodules may be so crowded together that practically no normal lung tissue can be seen. This condition is likely to occur in occupations, such as sandblasting, where there has been exposure to dust containing a very high proportion of free silica. Histologically, the silicotic nodule is a mass of concentrically laminated dense fibrous tissue.

Rounded or oval areas of *massive fibrosis* may be found centrally or peripherally situated in the lung, usually about the middle zones. The masses are nearly always bilateral and may be multiple, and frequently more extensive in one lung. In these cases

the remaining parts of the lungs usually show discrete nodules. It is possible that the massive fibrosis is inflammatory in origin. There is a second form of distribution met with following exposure to very high concentrations of free silica dust. It is known as the *cuirass form.* In it there is a layer of fibrosis extending from the pleura to a distance of a centimetre or more into the lung substance, and frequently over its whole extent, and with nodules of denser fibrosis embedded in it.

The presence of tuberculosis may render the diagnosis of silicosis at necropsy very difficult. Either the tuberculosis may be completely obscured by the silicotic process, or the silicosis may be obscured by the changes produced by tuberculosis which may have left no unaltered silicotic tissue. In the first instance, the presence of tuberculosis may be determined only after the finding of tubercle bacilli in the lung or a positive result to animal inoculation. In the second instance, great difficulty may be found in establishing a diagnosis of silicosis.

Changes in the lymphatic glands are important in the diagnosis of silicosis. The tracheal and bronchial glands are hard and, on section, dark grey or black, often showing severe concentrically arranged systems of nodular fibrosis. Parts of the cut surface may show white points or bands of fibrous tissue, or the whole of the gland structure may be replaced by fibrous tissue. Some glands in a group may be more affected than others, and several stages in the replacement of the glandular tissue by nodules may be seen. The lymphatic glands may show foci of tuberculosis, while tuberculous lesions are not apparent in extensively silicotic lungs.

The Occupations in which Silicosis Occurs

In reviewing the occupations in which silicosis occurs it is found that in all of them the workers are exposed to the risk of inhalation of dust of uncombined silica, in the form of quartz, or the chalcedonic forms as flint, chert, and Tripoli, and occasionally in an amorphous form as in kieselguhr and Neuburg chalk. The free silica may be the only constituent of the dust, as, for example, in sandblasting with quartzose sand and in crushing flint, or it may occur in the dust mixed with other substances, either natur-

ally like the quartz in granite, or artificially like the ground flint in earthenware pottery. The characteristics of the diseases which result from exposure to pure silica and mixed dusts vary in a way which appears to bear a definite relationship to the mineralogical composition of the dust.

(1) *The Sandstone Industry*

The sandstone quarrying, mining, and dressing industry represents perhaps the most widespread of all silicosis-producing industries in Great Britain. To a great extent, processes in cutting, shaping, dressing, and crushing of the stone are carried out near the places where it is got from the quarry or mine.

The sandstone-producing districts are the northern, central, and south-western counties of England and the south and east of Scotland. Chepstow Castle and Liverpool Cathedral are built of red sandstone, and Bristol Cathedral and Furness Abbey are part sandstone and part limestone. Early in the nineteenth century a good deal of Edinburgh was built in Craigleith sandstone, the supply of which is now exhausted. The famous pennant stone from the Forest of Dean is grey sandstone, that from Yorkshire is mostly brown, and a beautiful red sandstone is quarried in Corsehill, Dumfries. Other quarries are in or near Elgin, Alnwick, St Bees, Carlisle, Penrith, Lancaster, Bolton, Bacup, Darley Dale, and Matlock.

Sandstones are sedimentary siliceous rocks containing from 75 to 95 per cent of free silica and consisting more or less of quartz grains mixed with other minerals, and held together by a cement of varying composition and proportion. The composition, amount, and hardness of the cement are important factors in determining the dangerous characters of the dust produced. In Great Britain about 12,000 men are employed on the processes in quarries, and in addition there are about 20,000 sandstone masons employed in builders' and sculptors' yards throughout the country.

The workers include rock-getters who work at the stone face in quarry or mine; quarrymen or stone cutters who rough-hew the blocks; masons who shape and carve the stone to dimensions and patterns; planers, sawyers, and turners who operate stone-

cutting machines; drillers with pneumatic hand tools; crushermen, labourers, and cranemen; and builders, fixers, wallers or wallstone dressers who frequently do some dressing of the stone. The action of wind has the most important beneficial influence for workers employed in the open. Unfortunately, the worker is not always able to stand to windward of the point of origin of the dust produced by neighbouring workers.

The stonemason working in closed or partly closed sheds is liable to be exposed to dust produced by his neighbours as well as by his own work. Danger is increased by the practice of brushing the dry dust and debris from the surface of the stone and by blowing with the mouth while carving. Wetting the surface of the stone by rain has some influence in diminishing dust, more especially in getting the stone from the quarry, but it has little effect in reducing the fine dust given off by the action of a cutting tool.

Stone-crushing plants are frequently found in quarry sites for using up rubble to make road material. At the crushers, elevators, and screens, dense clouds of dust are frequently given off and travel for considerable distances, so that though few workers may be employed on the crusher-house plant, many may be subjected to the dust produced by it.

The workers most affected are the stone masons, and after them the quarrymen, rock-getters, planers, and wallstone dressers. Tunnel miners and grave-diggers working in sandstone may be affected too. The disease is more common after 40 years of age, and after 20 years in the industry.

(2) *The Granite Industry*

The granite industry assumed importance in Great Britain after the destruction by fire of the wooden town of Aberdeen in 1741. The present Granite City was planned and gradually built up of stones fashioned in the local quarries. Although the art of polishing granite stones for ornamental purposes was carried out in ancient Egypt, it became a lost art until it reappeared in Scotland about 1820. Since that date the city of Aberdeen has become the centre of the monumental granite trade, and the building and monumental sections of the industry have flourished side by side. Today, more than 80 per cent of the granite used for the manu-

facture of monuments in Aberdeen is imported from Norway, Sweden, and Finland.

Granites and allied rocks of igneous origin are found in many parts of the world. They are characterized by a crystalline structure and a certain hardness or toughness which demands special methods for quarrying them and adapting them for use. True granites are found in the west and north of Scotland and in Cornwall, Devon, and the Lake District of England. Other igneous rocks resembling granite are distributed in many parts of Great Britain. True granite consists of orthoclase feldspar, quartz, and mica. The chemical composition is distinctly acid, there being from 65 to 75 per cent of silica. In intermediate rocks – for example, the syenites and diorites – the silica percentage varies from 55 to 60; while in the basic rocks, dolorite and gabbro, the silica content is from 45 to 55 per cent.

The occupations in the granite industry may be classified as follows:

(i) *Labourers* are unskilled workers employed in removing overburden, or loading and filling granite, and they may assist in blocking, getting, or drilling.

(ii) *Getters* are skilled quarrymen who get the granite from the quarry face and roughly square it into blocks of suitable size. The group includes blockers and rockmen.

(iii) *Drillers* include workmen employed in using all forms of drills, including hand drills, steam drills, and wet and dry air drills.

(iv) *Settmakers* shape the setts or stone blocks for road material by means of hand hammers. They work in a shelter or open shed.

(v) *Kerb-dressers* are sometimes referred to as masons, particularly in Leicestershire.

(vi) *Crushermen* include all workmen employed in crushing mills and in concrete works. The group includes breakermen, screenmen, oilers, labourers about the mill, and loaders.

(vii) *Building masons* are skilled workmen engaged in cutting and dressing granite in builders' yards. Some of these work only with hand tools, and others use pneumatic tools. The use of the pneumatic tool varies considerably in different districts. In Cornwall it is used to a comparatively slight extent. In Aberdeen the

pneumatic tool was found to be more in evidence among building masons. These pneumatic tools may be for cutting or surfacing. Building masons work in open sheds.

(viii) *Monumental operatives* or monumental masons. This group includes squarers, duntermen, finishers, and turners working in monumental yards. The squarer uses a hand chisel and a pneumatic cutter. The dunterman is employed in operating the pneumatic dunter or surfacing machine (Plate 9). The finishers use the pneumatic tool almost exclusively for their work. Turners work with a power-driven lathe. The squarers can work in a shed by themselves or in the same shed as finishers. The duntermen work in widely open sheds in the yard. Turners are, as a rule, in the same shed as the polishers.

(ix) *Polishers* are employed in monumental yards. Polishing is a wet process; it may be done by hand but, as a rule, machinery is used. Where comparisons have been made between sandstone and granite masons it has been found that granite dust is less prone to cause silicosis than is sandstone dust. In one series examined the proportion of cases of silicosis in sandstone workers was 42 per cent of those radiologically examined, and 17 per cent in the case of the granite workers.

(3) *The Pottery Industry*

The Potteries of Staffordshire began before there were canals or railways, and an export trade was established using packhorses and mules for the transport of raw materials and finished goods. Today the North Staffordshire Potteries consist of the towns of Tunstall, Burslem, Hanley, Stoke, Fenton, and Longton, federated in the County Borough of Stoke-on-Trent. Before 1939 they gave work to 67,000 persons, of whom 55 per cent were women and girls. Among these, nearly 35,000 were employed in general earthenware, more than 10,000 in china, just over 18,000 in tiles, nearly 6000 in sanitary earthenware and sanitary fireclay, about 4000 in electrical porcelain, and just over 2000 in Rockingham and jet.

In 1720 John Astbury introduced finely powdered calcined flint into the body of his chinaware so as to produce on firing a remarkably white and hard product. In 1865, in her *Life of*

Josiah Wedgwood, Eliza Meteyard states that at first the flint, being used but sparingly, was kept in cellars and private rooms, and when reduced to powder in large iron mortars it was passed through hair sieves. This process was slow and extremely injurious as, in spite of every precaution, the dust was inhaled by the workmen and produced lung diseases of various kinds. She relates in detail how a painter employed in the potteries was struck by overhearing the story of the fatal effects of the flint. It occurred to him that the crushing could be performed by millstones under water, and the adoption of this method led to a considerable reduction in the amount of dust. Unfortunately, in the manufacture of ceramics in certain circumstances the dry method for the milling of flint is still employed.

The industry includes several distinct branches, subdivision being determined by the nature of the article being manufactured and the materials entering into the composition of the ware. The principal divisions are: earthenware, tiles, sanitary earthenware, and electrical fittings; china; jet and Rockingham; sanitary fireclay; stone-ware; and coarse ware. The occurrence of silicosis is especially associated with the first two groups – namely, earthenware and china – and the others will not be further considered.

In the manufacture of earthenware, the ingredients – ball clay, china clay, china stone, feldspar, and flint in a ground state – are mixed together in the form of liquid slip, from which excess water is removed by pressing. The resulting composite body is made into the consistence convenient for the manufacture of the various articles. So long as it remains moist it is harmless, but in the process of manufacture fragments fall on benches and floors or adhere to the clothing of the workers, become dry, and give rise to dust. After the article has been made in the plastic form, partly dried, it is frequently smoothed on a revolving disc and fettled, to remove irregularities of surface and edges. The articles are then placed in fire-clay saggars with some sand and fired in the oven. On removal from the oven the loosely adherent sand is brushed off. Subsequent processes of decoration and glazing are not associated with the occurrence of silicosis.

English china is made from calcined bone, china clay, and china stone, but it contains no added flint. The processes of

making the articles are similar, in general, to those employed for earthenware, but the articles require much more support in the process of firing than do earthenware articles, and to provide this support they are placed in finely powdered flint. The flint is prepared by calcining and subsequently crushing it in the dry state. When the saggars of china ware are taken from the oven, the flint, which was used in placing the ware, is removed by women in a series of processes known as scouring. Finally, after the ware is decorated and glazed, blemishes are removed by polishers, who frequently use finely powdered flint mixed with water as a finishing abrasive, on a rapidly revolving wheel.

It will be seen that the exposure to flint dust in the pottery industry occurs in the processes of the preparation of the flint by grinding; in the manufacture of general earthenware, earthenware tiles, sanitary earthenware, and electrical fittings, amongst those manipulating the earthenware body in the plastic and semi-dry states; in the manufacture of china, those employed in placing the biscuit ware in powdered flint and in removing the flint from it after firing; and in polishing the finished ware with the use of flint. It is in those processes that the highest incidence of silicosis is found to occur.

(4) *Tin Mining*

Objects made of tin have often been found in tombs of Ancient Egypt. At some early date the Phoenicians obtained tin from England. Pliny stated that it came from the *Insulae Cassiterides*. In other words the Romans went to the tin islands to exploit the Cornish mines. *Tinstone* or *cassiterite* is the sole source of commercial tin. It is stannic oxide contaminated with copper pyrites, arsenical pyrites, and other metallic sulphides. The fortuitous association of tin and copper minerals in parts of Cornwall led to the manufacture of bronze by the Romans at an early date.

In the Cornish tin mines the mother rock is granite. Before 1904 there was a high mortality from respiratory disease among rock-drillers in the mines. Modern methods of dust suppression have greatly reduced the incidence of silicosis in this industry. The dust risk is greatest in the operations of drilling, shot-firing,

and the shovelling of ore into trams. Although dust may be found lying thick upon ledges in the walls of the stope cavities, the amount of this redispersed into the air is probably insignificant save at the time of shot-firing. The disturbing of dust on the floor of levels by the feet of the men as they move about does not occur in the Cornish mines owing to the constantly wet state of the floor. When work has been done in an improper manner the atmosphere is comparable in appearance to that in a bathroom full of water-vapour. The measures employed to combat dust are natural ventilation, wet rock-drilling, mist-projectors and hand-operated water-sprays for directing water on the piles of broken ore before and during shovelling.

The work is done in 2 shifts daily and shot-firing is done only at the end of a shift. During shot-firing all the men leave the mine, with the exception of the drillers, who withdraw to a safe distance upstream in the air-current, and wait to count their shots as they explode. Having done this they also leave the mine which is then blown through with compressed air for not less than 2 hours and remains empty for not less than 3 hours. The familiarity of the skilled miner with dust is apt to result in carelessness. The penalty for offence, moreover, has not the immediate and dramatic quality which breach of the regulations is apt to produce in a coal mine, and close supervision of the miners is difficult because they work in pairs in widely scattered working-places. It is desirable that education in this respect should be authoritative, imaginatively planned, and continuous through the man's working life.

(5) *Haematite Iron-ore Mining*

Ferric oxide is widely distributed in nature as *red haematite*. Compact massive forms occur and include a reniform variety known as *kidney ore*. Much haematite occurs in an earthy form, when it is termed soft red ore. The term *brown haematite* covers a class of hydrated oxides. As we have already seen (p. 177), the effects on the lungs of the inhalation of iron-oxide dusts are innocent. But the case for the haematite miner is different because he has to blast and excavate haematite deposited in detrital quartz.

Haematite mining has been carried on in Cumberland since the Roman occupation. With the introduction of the dry pneumatic drill in 1913 the incidence of silicosis rapidly increased. During the 6-year period, 1930 to 1935, 48 haematite miners were certified in England as having died from silicosis. No case was certified during the year 1930, and the numbers for the subsequent years were 10, 7, 6, 13, and 12 respectively. Until about 1930 silicosis had not been generally regarded as an occupational risk in this industry. At that time it was pointed out that the Cumberland and Lancashire group of iron-ore miners had a higher mortality rate than the rest of the ironstone group for phthisis and other respiratory diseases, while the maximum incidence for phthisis was at a later period. Since that time a form of pulmonary fibrosis, frequently accompanied by tuberculosis, has been proved to exist in this industry. The change in the outlook may have been brought about by a change in methods of working. About 1913 hand-drilling was replaced by machine-drilling and with it more frequent blasting was made possible.

The dust to which haematite iron-ore miners are exposed differs from that with which other workers come into contact who suffer from silicosis, and in view of this important point in the aetiology of the disease, it may be well to consider certain geological data. Much of the haematite deposits of Great Britain are precipitates from ferruginous solutions which must have been in most cases charged with carbon dioxide, and such solutions would carry dissolved silica, which would be simultaneously precipitated with the iron, generally in the form of finely divided quartz. Also, in cases where the calcium carbonate of calcareous rocks has been replaced by iron compounds, all the insoluble siliceous constituents of the original calcareous rock will find their place in the newly formed iron ore.

Thus a siliceous limestone will give place to a siliceous iron-ore, in which the silica is mainly in the form of detrital quartz. Further, quartzose rocks may form one side of a lode of ore, and these have often to be blasted and cut in order to work the ore-deposit. Insoluble residue of better-class ores amounts to 5 or 7 per cent, but rises to 15 per cent, mostly in the form of free silica,

and in lower-grade ores the insoluble residue and free silica may rise to much higher figures. Some ore bodies may have definitely siliceous courses and the bounding wall of a lodge may be highly siliceous.

In the Cumberland mines from 1913 to 1935 the atmosphere underground became heavily polluted with haematite dust. The number of particles sometimes reached 4–5 millions per ml. Improved ventilation was introduced together with the Wetherill apparatus, a mist-projector producing a vapour-mist of minute particles of water containing one per cent of castor oil. In operation the holes in the rock face are first charged with explosive, the shot-firer then lights the fuses and on the way out he turns on the compressed air to set the atomizer in operation.

The new regime was introduced in 1935. Dust allaying by the mist-projector diminished the dust content of the air to an average of 2500 particles per ml. Medical control consists in selecting only fit men for work, radiographs of the chest being taken before admission and periodically thereafter. Uncomplicated pneumoconiosis or x-ray reticulation is not in itself an incapacitating condition. The single disabling and death-causing factor is infection, usually tuberculosis. Elimination of infected miners together with proper selection of new workers, and engineering control, have led to very great improvement.

(6) *Coal Mining*

In 1920 the toll of silicosis among men driving hard-headings in South Wales coal mines was first recorded. Almost simultaneously the doctors of Radstock in the Somerset coalfield noticed a chronic lung disease in miners employed in *branching*. This work involves machine drilling in driving roads through highly siliceous sandstones called Pennant rock, grit, or flag, and known locally as *greys*. In 1924 one of these men died and the lungs at necropsy showed silicosis. In 1925, 12 of these rock-drillers were examined by x-rays; all but one were found to be suffering from silicosis, the one case being doubtful. In 1946 a local study of the problem confirmed the serious incidence of silicosis among Somerset miners engaged in drilling and blasting in Pennant rock.

Only a small percentage of coal miners develop classical silicosis. The processes underground in a coal mine which may involve exposure to silica dust are:

(i) *Ripping:* taking down the roof or top of a roadway, to make height.

(ii) *Brushing:* ripping or blasting of the roof and using the debris for building stone packs.

(iii) *Driving a hard-heading:* which is a drift, tunnel, or roadway driven in rock or through hard measures.

(iv) *Driving a cross-measure drift:* which is driving a roadway in such a direction as is necessary to form a travelling road from stratum to stratum.

More than 60 names have been given to variants of this job. Hand drills and compressed-air percussive drills are used. The dusts met with in these processes are mainly those evolved from clift, bind, or shale, which may contain as much as from 40 to 60 per cent free silica and rock, bastard rock, or sandstone, in which the free silica may range from 60 to 85 per cent.

(7) *The Slate Quarrying and Dressing Industry*

This industry is concerned with quarrying or mining the rock, and making and shaping slates for roofing and other structural work. The large blocks of slate are conveyed to the mills, where they are sawn to sizes with power-driven saws cutting across the grain. The blocks are then split with chisel and mallet and cut to required sizes by hand or machine. Slate is the typical cleaved rock. The most important constituents quantitatively are silica, free as quartz, and combined as silicates of aluminium, iron, alkalis, and other bases. In Penrhyn slates quartz exists to the extent of from 35 to 43 per cent, while the total silica (free and combined) is from 58 to 63 per cent. Silicosis in slate-workers is sometimes referred to as slate-workers' lung or schistosis.

(8) *The Grinding of Metals*

The processes which cause silicosis in the grinding of metals are confined to those in which grindstones composed of natural sandstone are used. The industries in which metal-grinding is an important process can be divided broadly into (i) the cutlery and

edge-tool trades, and (ii) the machinery, general engineering, and foundry works.

Grinding may be done wet or dry and the metal being ground may be moved by hand or by mechanical power on a machine. In wet grinding, water is laid on from a tap or spray above the stone and is drained away beneath. Wet grinding is done by machines, especially in the manufacture of files, saws, and machine-knives, when the metal being ground is fixed to a part of the machine which moves under or across the revolving grindstone. The processes of dressing the grinding surface of the grindstone are very important from the health point of view, on account of the high concentrations of dust given off. Dust is produced by attrition of the grindstone in all processes in the grinding of metal and it varies in amount with the consistency of the stone, the hardness of the metal, the shape of the article being ground, and the amount of force exerted.

The grinding of metals on sandstone wheels has been recognized for generations as a cause of silicosis and an increased mortality from tuberculosis. A decline in the number of deaths has occurred from 2 causes: (i) the change from sandstone to artificial abrasive wheels, and (ii) the effect of stringent preventive measures in the grinding-rooms. At the coming into force of the Workmen's Compensation (Silicosis) Scheme for the grinding of metals industry on 1 July 1927, there was a widespread departure from the use of sandstone wheels and a corresponding adoption of the artificial abrasive. Artificial abrasive wheels are composed of carborundum (silicon carbide) or some form of aluminium oxide, and contain only a small proportion of silica or none at all.

(9) *Iron and Steel Foundries*

The founding of metal is an ancient craft, so ancient that, under the weight of tradition, both employers and workers have regarded the hot, dusty, and dangerous conditions as inevitable; and industrial countries did not campaign for better working conditions until about 1930. Metal castings vary in weight from less than an ounce to many tons. There are therefore widely varying types of foundries, known as light, heavy, and jobbing

foundries. Some of these are mechanized. In Great Britain steel foundries are concentrated in Sheffield, Lancashire, Middlesbrough, South Wales, and on the Clyde. Iron casting is mainly done in Falkirk, Birmingham, and the Black Country. Foundries in the South of England handle chiefly non-ferrous metals. Foundry work employs large numbers of men. Its importance is seen in the motto adopted by the University of Birmingham Metallurgical Society: *The hand that wields the ladle rules the world.*

Founding depends on the construction of complicated sand moulds which, with careful treatment, can be inverted and will stand up to the stream of liquid metal without being distorted. Sand used for moulding may be *naturally bonded*, in which case it holds together because it contains clay. *Synthetic moulding sands* are mixtures of silica sand with a *binder* such as ball clay, china clay, or dextrin. In founding a *pattern* of the casting is first made usually in wood. A mould is then made by ramming sand round the pattern so that when this is removed an impression of it remains in the sand. Molten metal is then poured into this impression and when it cools it forms a solid casting with the same shape as the pattern. If a hollow casting is required a sand *core* is prepared with its external surface conforming to the internal shape desired in the casting. The core is then placed in the impression left in the mould by the pattern so that the molten metal fills the space between the core and the mould. These cores are frequently made of oil-bonded sand and are baked to give them strength and rigidity. A complicated large mould may have over a hundred loose core pieces and take many days to make.

Moulding sand used in foundries is generally excavated from quarries. There are excellent supplies of naturally bonded sand near Mansfield, Wolverhampton, Erith, and Leighton Buzzard. The sand mixtures for moulds or cores are prepared either by hand or in a special sand-preparation plant. The *moulders* make the moulds on a bench if the castings are small, on the foundry floor if they are medium size, and in a pit if they are very big. The moulding of the sand is done in a pair of *moulding boxes* which are rigid frames, generally of cast iron, for carrying and

supporting the sand. A channel or *runner* must be cut through the sand in the one half of the mould so that liquid metal can be poured down this runner and flow into the cavity. One or more channels called *risers* must also be made, so that the cast metal fills the cavity in the mould and rises up the riser which also provides an outlet for gases. When the metal has solidified and the two halves of the mould have been separated, the casting remains attached to a neck of metal representing the runner, which is subsequently cut off. After the metal in the moulds has been allowed to cool, the moulding sand and cores are knocked or shaken away from the casting by the *knock-out*, the dustiest of all foundry operations.

In particular cases the moulder, as a final treatment, dusts the mould with a parting-powder which may consist of silica flour. A bench moulder may make as many as 150 boxes in a shift, and because the parting-powder may be dusted twice on each box he may be enveloped during the shift in 300 dust clouds each having a high free silica content. The most important forms of silica used for parting-powders are Tripoli and Neuberg chalk. Tripoli is generally regarded as a chalcedonic variety of silica, and contains about 97 per cent of silica. The substance generally known by the name is imported from Seneca, Missouri, and is of two kinds, which differ in their mode of origin but resemble each other in composition. The name comes from Tripoli in North Africa, where the product is a true diatomite. Silicosis with tuberculosis has occurred from exposure to dust of Tripoli in England, in men who had been employed for periods of 10 years or more mixing ingredients in the manufacture of parting-sand. In foundries in the United States of America as late as 1930, one could see a silica wash being used to face the moulds in steel foundries and observe this wash being applied with a spray gun. All persons in the immediate vicinity were then showered with a mist, of which the essential ingredients were water and fine silica made from ground flint, one of the most deadly silicosis producers known.

In foundry work *fettling* or *dressing* means cleaning. The term *chipping* is applied to the use of tools to remove rough edges of metal from castings. The removal of adherent moulding

sand and core sand is called *stripping*, *roughing-off*, or *mucking-off*; it is done with hand tools or portable pneumatic tools. Small castings are cleaned and smoothed by *rumbling* in a revolving barrel in which they are tumbled against each other and some abrading agent. Abrasive blasting is carried out in chambers or cabinets making use of *sandblasting* or *shotblasting* by compressed air or of the *wheel abrading machine*. After the castings are blasted they are sent to the fettlers or dressers, who use *pneumatic chisels* as well as hammers, chisels, and brushes to remove the burnt-on moulding sand and the rough edges of metal. Some castings are smoothed by *grinding* with *abrasive wheels* of carborundum, emery, or alumina. In special cases the *hydroblast* is used for cleaning castings.

The main dust-producing processes in foundry work are those connected with the removal of the moulds and core sands from castings and their subsequent cleaning and dressing. Moulding and core-making are not particularly dusty jobs, because the moulding materials are damp when handled. The preparation of sand, the application of parting-powders, and the cleaning of moulds and metal plates with jets of compressed air are all dusty jobs. The knock-out of castings and the dismantling and rebuilding of furnace linings and ladles also produce much dust. But the jobs in the fettling shop cause the finest dust, so that fettling work with pneumatic chisels, sandblasting, shotblasting, wheelabrator work, portable-grinder work, rumbling, and stripping are all dangerous jobs so far as dust diseases of the lungs are concerned.

The first large-scale investigation into the chest condition of foundry workers in England was completed in 1923. During a general enquiry directed especially into the health of metal grinders using sandstone wheels, dressers of steel castings were examined and it was found that 22·8 per cent had pulmonary fibrosis. In comparison, 73 per cent of a group of 495 wet sandstone grinders showed pulmonary fibrosis; the fettling of castings was therefore shown to be a less unhealthy job. It should be noted that at this time fettling was done mainly with hand tools; pneumatic tools came into more general use later. When the silicosis compensation schemes came to be formulated, steel

dressers, but not iron dressers, were scheduled as a group entitled to compensation for silicosis.

There are three reasons why the incidence of dust diseases of the lungs is higher in steel than in iron-foundry workers. The melting point of steel is approximately 1600° C. as opposed to 1100° C. for iron. Therefore there is a greater tendency for the metal to penetrate the sand mould and cause *burning-on* of the moulding-sand on steel than on iron castings. The *burnt-on* sand is difficult to remove and pneumatic chisels are used to clean steel castings, whereas hand tools usually suffice in iron-fettling shops. The pneumatic tool breaks up the sand granules into freshly fractured small particles which, when inhaled, are more active in producing silicosis. Finally, the mixtures used for making moulds for steel castings contain more free silica than moulds for iron castings. Steel moulding-sand contains up to 99 per cent of free silica, whereas iron moulding-sands rarely have more than 80 per cent and usually have less.

Until 1943 the known cases of pneumoconiosis in iron-foundry workers were few, and they were not at first included in the compensation schemes. The difficulty about bringing them under such a scheme was partly due to the fact that hitherto the classical silicotic nodule had dominated the study of the pathology of the industrial diseases of the lungs. Any case in which classical silicotic lesions were not found could not be diagnosed as silicosis and hence was not eligible for compensation. But the inhalation of mixed dusts containing free silica and other dusts such as iron oxide tends to retard the development of classical silicosis. New pathological studies culminating in the work of McLaughlin and others in 1950 showed that the main lesion in the lungs of iron-foundry workers, particularly iron fettlers or dressers, was another type of nodule with a linear and radial pattern as opposed to the whorled arrangement of fibres in the classical silicotic nodule. To this type of lesion Harding, Gloyne, and McLaughlin applied the term *mixed-dust pneumoconiosis* or *mixed-dust fibrosis*. The survey directed by Dr A. I. G. McLaughlin, H.M. Medical Inspector of Factories, was a concerted effort by employers, trade unions, Government departments, statisticians, pathologists, clinicians, and engineers. The investigation included the

results of clinical and radiographic examinations of 3059 workers in 19 foundries, an analysis of the records of lung disease in foundry workers in the files of the Factory Department and the Silicosis Medical Board, pathological investigations of the lungs of 64 foundry workers, and dust surveys in 3 foundries. As a result of this work, under the *Iron and Steel Foundries Regulations, 1953*, which became fully operative in 1956, iron-foundry workers are protected by special regulations and paid benefit if they develop pneumoconiosis.

(10) *Amorphous Forms of Silica*

We have seen that amorphous forms of silica are used as parting-powders in foundry work, but in addition to this they are used as polishing compositions and also in the manufacture of steel. Neuberg chalk is a natural deposit of siliceous material occurring at Neuberg on the Danube. The composition varies somewhat in different localities and the silica content is sometimes given as from 65 to 86 per cent. Besides free silica it contains from 7 to 8 per cent kaolin, and traces of iron and magnesium. There are indications that silicosis has followed exposure to the dust. In one factory 6 of the men employed on a process in which this material was used for periods from 3½ to 17 years had died of tuberculosis or silicosis with tuberculosis. Following this experience, examinations were made and early signs of silicosis were found in three men. In another factory one or two of the men who had been employed 10 and 12 years on the process showed early signs of silicosis.

Diatomite (kieselguhr, diatomaceous earth) is composed of minute siliceous skeletons of aquatic plants of marine or freshwater origin. When pure it contains up to 96 per cent of silica, but it may contain alumina, iron, lime, magnesia, and alkalis, reducing the silica content to about 75 per cent. It is used as a mild abrasive or polishing medium, as filtering material, and for insulating purposes. Where men handled a diatomaceous deposit in Santa Barbara County, California, which yielded, when dry, a silica content of 85 per cent, much dust was produced in conveying, grinding, drying, and bagging. Clinical and radiological examinations were made of 108 men, and silicosis was found in

81 (68·5 per cent); very early in 15, early in 45, moderately advanced in 15, and advanced in 6.

In a process in the manufacture of steel, in which a number of men had been exposed to dust of keiselguhr for 6 years, in Great Britain, radiographic examination showed more fibrosis than usual, with very early indications of a modified form of nodulation in some of them.

(11) *Flint Crushing*

Flint, a chalcedonic form of silica, is crushed for making chicken grit, sandblasting grit, abrasive papers, and brake-sand for tramcars. The flints may be first calcined, in order to make the material more friable and whiter, before crushing. High concentrations of dust are produced at the crushing and screening processes and it is an extremely difficult problem to control and dispose of it. Sand becomes dangerous when it is dried and manipulated in such as way as to give rise to fine particles of quartz dust. This occurs in sieving dried sand for sandblasting, for facing bricks, in the manufacture of glass and other products, and in mixing dried sand with other substances.

(12) *Silica Milling and the Manufacture of Abrasive Soaps*

These processes may be carried on in the same factory, but more usually silica milling is done in one factory and the ground material is supplied to manufacturers of abrasive soaps. The silica may be quartzite rock containing over 90 per cent of silica, or crystalline quartz, or quartzsand. It is dried, crushed, and ground to a fine powder by machinery, which is totally enclosed and provided with exhaust draught to remove dust. In the manufacture of abrasive soaps, ground silica is mixed with powdered soap and anhydrous sodium carbonate. Dust is produced in the processes of mixing, sieving, and packing the powders. About 1928, numerous firms who had no knowledge of the dangers of exposure to the dust and were inadequately provided with protective measures became engaged in this manufacture. This resulted in the occurrence at one factory of three or four cases of respiratory disease which was regarded as

an acute form of silicosis. The manufacture began in England in 1910, but was not important in quantity until 1921, and in 1928 it was given up owing to the death of workers attributed to the inhalation of the dust.

(13) *Refractory Products*

This industry has a special interest in relation to silicosis, because it was the first to which the Workmen's Compensation (Silicosis) Act, 1919, was applied. It is engaged in the manufacture of refractory materials which contain over 80 per cent of total silica and are used in the construction of furnaces, flues, and crucibles.

It is a small industry; some 3000 workers are employed in Britain. The processes include the quarrying or mining of the raw material, usually a sandstone of the coal measures called ganister, which occurs in open quarries and in mines, or, in some localities, sands or pocket-clays are used. The raw material, which contains usually from 92 to 98 per cent of silica, is crushed, mixed with suitable bonding substances, and made into silica bricks, silica cement, steel-moulders' composition, and similar products for use as refractories in the manufacture of metals, especially steel or steel castings, and in the construction of gas-retorts and flues. Mechanization is improving conditions in the manufacturing processes from the health point of view, especially with the use of continuous kilns which the workers do not enter.

(14) *Sandblasting*

This is the process of projecting sand or other grit, by means of compressed air or steam, or by the centrifugal action of a wheel against a surface. It was introduced about 1904 and is used:

(i) In metal works to remove adherent sand and irregularities from castings.

(ii) To produce, on clean metal, a surface suitable for subsequent treatment by coating with enamel or another metal.

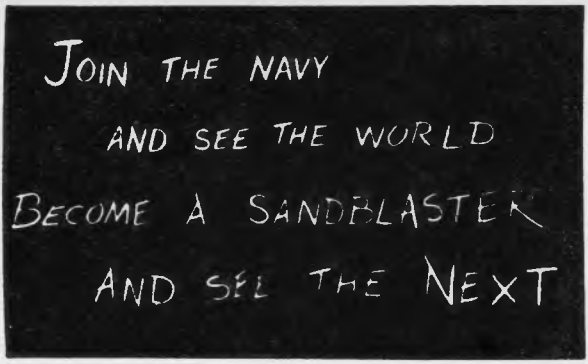
(iii) For etching glass and treating other non-metallic articles.

(iv) For removing paint from, and otherwise cleaning, large surfaces, as of ships and buildings.

Sandblasting of metal articles can be carried out in various ways. For large articles the operator works in a specially constructed room, and he is protected by special clothing and a helmet which is supplied with pure air under pressure (Plate 10). The dusty air is removed from the room by exhaust draught. Smaller articles are treated in a closed cabinet, the worker passing his arms through guarded holes to direct the abrasive while watching the process through a glass panel. Other types of apparatus are the mechanical turntable and revolving barrel, in which the sandblast is directed from a fixed point and close application of the worker is not necessary. Wheel-abrading machines vary in design, but in all of them a rapidly revolving wheel projects the abrasive against the casting. Such machines are operated outside the blasting enclosure.

The dust hazard is from the abrasive when this consists of quartzose sand, crushed flint, quartz, quartzite, or granite. On hygienic grounds, metal grit and certain other non-siliceous abrasives have now replaced the siliceous material to a considerable extent, and where these are used on clean metal no siliceous dust is produced; when they are used for cleaning metal castings, however, dust is produced from the adherent moulding sand, which is highly siliceous.

There is no doubt that sandblasting, and to a lesser extent shot-blasting, is a dangerous occupation if adequate precautions are not taken. Risks are incurred by workers by faulty or careless methods, or through defects in the appliances, which require constant attention for maintenance. Silicosis is found amongst sandblasters in various parts of the world, and the same features, namely, a short period of employment and rapid course of the disease, characterize all the cases. In 1936 a survey was made of the silicosis risk in sand- and shotblasters in Great Britain. It was shown statistically that the average duration of employment of sandblasters who ultimately died of silicosis was 10·3 years, as compared with 40·1 years, which was the average duration of employment of all cases of silicosis irrespective of the causative occupation. Of course the men themselves are aware of that hazard. Thus in 1934 in a works in Coventry they chalked up the following notice –



PREVENTION OF SILICOSIS

In order to be certain that people will not get silicosis, it is clear that harmful dust must not get into the air which they breathe. But this presents great difficulties, because many factory processes and most types of mining are inherently dusty. Nevertheless, dust clouds *can* be controlled, sometimes partially and sometimes completely.

All silicosis is man-made. On the subject of its prevention there is no room for complacency, because the figures for deaths are going up every year. Thus in 1957 in Switzerland at an altitude of 7500 ft. in the Alps of the Canton Valais quartz dust was still producing silicosis amongst tunnellers working on the vast hydro-electric scheme known as La Grande Dixence. While it is true that dust suppression by wet rock-drilling was applied in the larger tunnels there were many small tunnels where all the drilling was done dry. *Why?* Things like this still happen in spite of the fact that at no time in history has more attention been given to the problem by doctors, engineers, chemists, physicists, and others; by organizations and individuals; and by employers and trade unions. The practical details of dust control are largely in the domain of the engineers. But the problem is primarily one of health, and it is the business and the duty of doctors to lead the campaign and to inspire the engineers, if necessary, to greater efforts.

Not only can dust be controlled, but in some instances the process can be altered so that no dust is formed. Again, it is possible, occasionally, to replace the substance which gives rise to the dangerous dust by another which is non-toxic or at least not so harmful. But in this event the process will still be dusty if dust control is not applied. There are some jobs in which it has not been possible, up to the present time, to control the dust, and in these it is necessary for the workers to be protected by dust respirators or by breathing apparatus. The basic principles of the prevention of silicosis can be considered under the four headings: (i) replacement of the harmful material by a less toxic one; (ii) dust suppression or control; (iii) personal protection of the worker; and (iv) medical examination.

(1) *Replacement or Substitution*

Though the principle of replacing a harmful material by a less dangerous one is of the first importance, it is not always practicable to put into operation. For instance, coal gives rise to enormous amounts of dust, but is indispensable. But when the material giving rise to a dangerous dust can be replaced, then the danger can be eliminated. Thus limestone has replaced kieselguhr for use in the slow cooling of steel ingots, and zircon can be used instead of silica flour for mould paints in foundries.

For building stones it is not always possible to substitute limestone for sandstone. The limestone of the Mediterranean basin was used to build the pyramids of Egypt, and builders and sculptors in stone have made use of it ever since. The chief sources of ornamental limestones and dolomites in Great Britain lie in a belt which crosses England obliquely from the Dorset coast, through Somerset, Gloucester, Northamptonshire, Rutland, Nottingham, and Lincolnshire, to the coast of Yorkshire. The drilling, blasting, shaping, carving, and crushing of limestone and dolomite are all harmless occupations. There is no dust hazard to men at work in limestone quarries, to stonemasons handling only limestone and dolomite, or to men who crush these materials to make stones. It follows that if more of our stone buildings were of limestone or dolomite instead of sandstone or granite, a lot of suffering from silicosis would be avoided. Unhappily, limestone

buildings cannot long withstand the acid atmosphere of industrial towns, which causes them to undergo weathering and to crumble away.

There are at least four industries in which the principle of replacement has been applied on a large scale – namely, flour milling, metal grinding, the pottery industry, and sandblasting.

The first of these is flour milling. Sometime about the year 1877 the introduction of steel rollers for milling corn removed the need for fashioning millstones from natural silica rock. The second is the grinding industry. For many years grinding wheels made of natural sandstone were used in the cutlery, edge tool, and other trades. In fact, the natural abrasives – hard, gritty materials, most of them siliceous rocks – have been used in various forms for sharpening, grinding, and polishing for 3500 years. These substances were at one time quarried in vast amounts – for example, Grindstone City, Michigan, in the years between 1835 and 1929 produced 25,000 tons of natural silica stones each year.

And then in 1891 Edward G. Acheson, of Monongahela, Pennsylvania, made the first synthetic abrasive, silicon carbide. Seeking to synthesize diamond, he was heating clay and coke in an electric arc. He obtained a product as hard as the natural abrasive corundum, and wrongly thought that what he had produced was a compound of carbon with corundum, hence *carborundum*, a misnomer adopted as a trade name for silicon carbide. It has a hardness of 13, compared with 15 for diamond and 10·5 for sandstone. The method of manufacture is simple; sand, coke, and sawdust being fused in the heat of the electric arc. Cheap hydro-electric power played its part, especially at Niagara. In 1899 fused alumina, or *synthetic emery*, was added to the manufactured abrasives. By 1945 the United States of America was making 30,000 tons a year of artificial abrasives.

Since about 1923 sandstone wheels have gradually been replaced by wheels made of carborundum, emery, or alumina, so that now sandstone is rarely used for any grinding or polishing job. Though it is not certain that the dust of silicon carbide is completely harmless, it is so much harder than sandstone that it gives off much less dust. The number of cases of silicosis among

grinders has been dropping year by year and, with the exception of those who grind castings on which there is burnt-on sand, here should be no cases of silicosis among grinders by about 1975.

Another instance in which it has been possible to replace a siliceous substance by a less harmful one is to be found in the pottery industry. In the placing of biscuit ware in the saggars so that they can be fired in ovens, it used to be the practice to embed the china articles in ground flint, which is nearly pure free silica. The incidence of silicosis amongst this group of pottery workers was high, but since about 1934 powdered alumina has been used to replace flint both for placing china ware and for polishing articles of pottery. Since then the number of cases has been getting less.

A fourth example of replacement is seen in the process known as abrasive blasting. In this job a stream of abrasive material is blown under high pressure at articles to clean them. It is used, for example, in the foundry industry to clean castings; it is also used to etch glass and to smooth the surfaces of metal before it is painted or enamelled. About 1904 sand was introduced as an abrasive, and though the number of workers was small, the incidence of silicosis and tuberculosis was high and the disease came on quickly. Since about 1920 sand for blasting has been almost entirely replaced by other abrasives such as steel shot and calcined alumina. The use of sand in blasting operations is now prohibited by the *Blasting (Castings and Other Articles) Special Regulations, 1949.*

(2) *Dust Suppression or Control*

Ventilation may be either local or general.

General ventilation is usually obtained by having open doors and windows, but this practice is not popular in cold weather and it puts up the cost of heating the workrooms. Air-conditioning is being adopted in some industries with good results. For this type of ventilation, in which filtered air is blown into the room and extracted by fans, the doors and windows should be kept closed. Where there are furnaces or ovens in a workshop, advantage is taken of the upward currents of air caused by the heat. In these rooms the incoming air is brought in low down in the walls

and the outgoing air is extracted through the roof. By contrast, in rooms in which there are no hot processes, the air currents are reversed. For example, in a room in which castings are cleaned, the air is blown in high up, either through the roof or the walls, and the air exits are placed low down.

When dangerous dust is given off, general ventilation is not enough because it allows the dust, even though it may be diluted, to get into the workers' breathing area. In these cases *local exhaust ventilation* should be installed. This consists of a hood, placed as near as possible to the source of the dust, and a duct leading from the hood to a fan which creates a current of air to draw away the dust. It may be taken to a dust collector or be discharged to the outside air. Exhaust ventilation has been applied to many industrial processes, but it is never really satisfactory unless it is combined with enclosure of the process. An example of this is the chemical fume cupboard, and the principle can be and has been adopted in many industrial processes with excellent results.

Experiments are now being made to avoid the use of the pneumatic tool for the fettling of castings. They include attempts to burn off the sand adhering to the castings with a very hot flame produced by acetylene, air, and a fluxing powder. Though iron oxide is given off in the fume, no harmful dust is evolved, and altogether it is a promising development. Another way to avoid the use of the pneumatic tool is to put the casting into an annealing furnace, in which a great deal of the burnt-on sand is removed from the casting. However, the fundamental method of tackling this problem is to control the technique of moulding and casting in such a way that the casting comes clean away from the mould so that the use of the pneumatic tool or any other method of cleaning is not necessary. In one foundry, where clean castings are the rule, there has been no case of silicosis for about 10 years. This is the ideal way of getting rid of a health risk, but so far it has not been possible to apply it generally.

If the dusty process can be isolated from other parts of the factory, the number of people exposed to the risk can be reduced. In some foundries all processes, from sand preparation and core-making to the final cleaning of the castings, are carried out in

one large workroom. The dangerous dust created in fettling becomes distributed through the workroom and affects to some degree all groups of workers. If the coremakers who handle damp sand work in a separate room, they inhale little or no dangerous dust. In general, the best policy is to isolate as far as possible all dusty processes, and to protect the workers engaged in them by good general and local ventilation together with personal protective methods. A less satisfactory way, but one which is better than nothing, is to operate a dusty process at night when the other workers have gone home. In some foundries the *knock-out*, that is stripping the castings away from the sand moulds, is done at night.

Water, foam, and oil may be used for allaying dust. In hard-rock mining, wet drilling is widely used. John G. Leyner, of Denver, Colorado, has been called the outstanding mechanical genius of rock drilling. In 1897 he brought to practical success the principle of the pneumatic-hammer drill. A blast of air was blown through a hollow drill-steel to remove rock cuttings from the hole. This produced so much dust that the drill became known as the *widow maker*, and miners refused to use it. In 1898 Leyner obtained a patent on the commingling of air and water in the drill-steel. This is the principle of the axial water feed and is the basis of all rock drilling (Plate 11). The adoption of the method had to wait upon the invention of suitable steel alloys, but by 1920 it was in use all over the world. As an engineering device it cools the drill and clears the chips of rock most efficiently from the hole: happily it also suppresses dust.

Where wet drilling proves impossible or objectionable foam-drilling equipment can be used. By means of compressed air a viscous foam is forced through the drill-steel from a closed vessel. Although the foam traps the dust, the method has the defect that when the bubbles burst they may release dust particles. Since the tungsten-carbide bit need not be cooled and will last for 300 feet of drilling as against 10 feet in the case of steel-alloy drills, a new drill called the *Holman dryductor* has been perfected which sucks the dust back through the axial channel in the drill steel and ejects it into a dust collector. Near the rock face after shot-firing a mist projector is used to allay dust. It is really an

atomizer using compressed air and spraying a mixture of castor oil and water in which the proportion of oil to water is about 1 in 100. After shot-firing nobody may enter the area until 8 hours have elapsed for the dust to settle.

An apparatus used to clean certain types of casting, and the safest yet devised, is known as the hydroblast. A high-velocity jet of sand and water is projected on to the castings, thereby removing moulding material, cores, and scale. The velocity of the water as it leaves the hydroblast gun is in excess of 3 miles per minute. It can be readily understood that this apparatus has greatly improved the cleaning of castings and much reduced the dust in the atmosphere of fettling shops where it has been installed. When it was first introduced it was hoped that it would take the burnt-on sand away from the castings and so avoid the use of dusty methods of fettling or cleaning. Unhappily, this hope has not been realized, because the cleaning has to be completed in the shotblast or wheelabrator chambers, or even with pneumatic tools.

New methods of dust control by water are devised from time to time. Thus the *First Report of the Joint Standing Committee on Conditions in Iron Foundries, 1956*, describes in particular a new type of wet decoring bar for use in foundry work. But although wet methods for dust suppression are a great deal better than nothing it should be pointed out that water is not as efficient as good exhaust ventilation in controlling dust. The finest particles are not brought down by water; indeed, they may actually be held in suspension by droplets in the air. Investigations have shown that in wet grinding, for instance, there may be more dust present in the air than when the grinding is done dry under efficient exhaust ventilation. If the damp dust is not removed, it will become dry and contaminate the air of the workroom. The addition to water of wetting agents which lower the surface tension of the droplets should theoretically help to engulf and control the dust particles more easily. They have been tried in some instances, but not with conspicuous success. The use of water is therefore not the ideal method of dust control and may give a false sense of security.

Dust particles can be removed from a space containing air or

gas by creating an electric field in it, thereby giving each particle a negative charge of electricity. The particles will then adhere to positively charged plates at the sides of the space. The method is used in Great Britain to clean certain chimney effluents and also coal gas, but it has not been applied to dusty factory processes except occasionally in dust collectors. In the gold mines of the Transvaal it is used successfully on a fairly large scale to control the air-borne dust. After a high initial capital outlay, the running costs are low.

Factories should be kept in a tidy state, and the floors, walls, and rafters should be cleaned regularly. Accumulations of dust on the rafters and ledges can be a source of air-borne dust, especially when there is vibrating machinery in the workroom. Dust should not be allowed to accumulate on the floor of the workroom, because it is easily kicked up to become air-borne. It should be removed daily by vacuum cleaning rather than by sweeping. Attention to these matters will reduce greatly the amount of dangerous dust in the air of the workroom.

(3) *Personal Protection of the Worker*

Where it is found impossible to control the dust completely, the worker must wear either a dust mask or an air-line breathing apparatus. A respirator must never be used as a lazy excuse for avoiding dust control.

A dust mask is a filtering device for removing particulate impurities from inspired air. It does not remove gaseous impurities, and its use by mistake as a gas mask could lead to a fatal result. The dust mask protects the nose and mouth. It is made of some light, durable material such as rubber, plastics, or aluminium. The edge of the facepiece is lined with soft rubber and may be fitted with renewable cloth facings. The filter medium may be paper, wool, cotton, wool-asbestos, glass wool, or various combinations of these substances. The filter is usually renewable. Dust respirators are most suitable for temporary use where there are short periods of exposure to dust, for example when asbestos dust collectors are being cleaned out. It is almost impossible for a man to do a heavy job continuously when breathing against the resistance of the filter of the respirator.

If an air line has to be used, it is better to have it attached to a helmet. This apparatus can be used to protect people who have to go into a dangerous atmosphere, whether the contaminant is dust, fume, or gas. A constant stream of warm clean air under slight positive pressure is blown into the top of the helmet, over the face, and out at the sides. This type of helmet is used by shotblasters, and if it is maintained properly and not taken off before the worker is out of the dangerous atmosphere, it gives excellent protection.

(4) *Medical Examination*

Medical examination is used in the prophylaxis of silicosis in two ways: the initial examination of applicants for employment in an occupation with a silicosis risk, and the periodical medical examination of workers engaged in such occupations. The object of the initial examination is to prevent workmen whose respiratory physique is defective, through malformation or disease, from entering the dangerous industry. The periodical examinations enable silicosis to be detected at an early stage so that the workmen concerned can be transferred to other work. The examinations are also a means of discovering men with pulmonary tuberculosis who not only expose themselves to additional risk by remaining in the dusty occupation, but are possible sources of infection to their fellow workers who, following exposure to siliceous dust, have been rendered more liable to develop tuberculosis. In addition, these examinations provide cumulative evidence of the changes in the workmen's condition. But x-ray examination does not prevent a man from contracting silicosis. It does help to get him out of a dusty industry before the damage to his lungs has gone too far. X-ray and other examinations can also indicate in which part of the job the danger lies, and as such they are valuable because greater attention can be given to dust control in the dangerous job. It is not fair to the workers, however, to use x-ray surveys deliberately in order to save the time and trouble of the engineers whose business it is to control dust clouds wherever they arise. Drs Edward L. Middleton and Andrew Meiklejohn have made notable contributions to this subject.

Pneumoconiosis of Coal Miners

We have seen (p. 190) that only a small percentage of coal miners develop classical silicosis, and that the mining occupations involving exposure to silica dust include ripping, brushing, driving a hard heading, and driving a cross-measure drift. But there is a very much commoner disease of coal miners working at the coal face resulting from the inhalation of a mixed dust of coal together with a relatively small proportion of siliceous material. This pneumoconiosis of coal workers occurs in all countries where coal is mined, and it is found also in trimmers of coal cargoes in ships. In the British Isles the distribution of the disease shows a preponderance in the South Wales coalfield. In Great Britain it accounts for more deaths than do all other forms of pneumoconiosis combined, and since 1930 it has constituted the greatest medical and social problem in all industry. We know that coal has been mined in England at least since the year 1234, yet 700 years were to elapse before doctors would accept the fact that coal dust *does* gain access to the lungs of the miner, producing a slowly progressive fatal disease. It must be admitted that medical men by their ill-informed complacency have a heavy load of responsibility to bear for this failure to discover the true state of affairs, a failure which constitutes what is probably the greatest disgrace in the history of British medicine.

For the years 1930–2 the Registrar-General's figures for the occupational mortality statistics showed that respiratory diseases were on the increase in coal miners; they were not benefiting from public-health measures to the same extent as other groups. By 1935 certifications among coal miners by the Silicosis Medical Board focused attention on the progressively mounting number of cases occurring almost entirely in South Wales, especially in the anthracite area around Swansea. In 1936 Dr Charles L. Sutherland, Chief Medical Officer of the Silicosis Medical Board, directed the attention of the Home Secretary and of the members of the Industrial Pulmonary Diseases Committee of the Medical Research Council to the fact that, in the previous 3 years, claims for compensation on account of silicosis by coal miners

had increased by 70 per cent, and these were almost entirely in South Wales. There was also a vast number of men whose cases were of anomalous types and who failed to obtain compensation.

The situation was a most unhappy one and called for immediate scientific investigation. In 1936 the Medical Research Council undertook to investigate the problem of chronic pulmonary disease among coal miners, with particular reference to conditions in the South Wales coalfield. The inquiry which was directed by Dr P. M. D'Arcy Hart extended over the years 1936–42 and consisted of a medical survey and pathological and environmental studies. The next step in the investigation of this complex problem was the establishment in Cardiff in 1945 of the Pneumoconiosis Research Unit under the direction of Dr Charles M. Fletcher. As a result of extensive researches Fletcher and his colleagues have made many useful contributions to the problem. The morbid anatomy of the subject has been investigated in detail by Professor Jethro Gough of Cardiff.

Coal-miners' pneumoconiosis presents three different clinical pictures. There is the pure coal-dust disease, non-progressive and benign unless focal emphysema develops, when it may be fatal. In this respect it is a more serious condition than the second disease, the classical non-infective silicosis of rock workers, in which focal emphysema is much less prominent. In coal miners these two diseases are often indistinguishably combined. Then there is a third disease process, due to the interaction between some infective process, often tuberculosis, and the dust ensnared in the lung. The fundamental similarity of coal-miners' pneumoconiosis to pure silicosis is close. It is possible that the differences may be due to some action of the coal dust in the lung in subduing the toxic effects of tuberculosis both generally and locally so that it is not clinically apparent, and so that the bacilli only occasionally escape from their fibrous prison to spread throughout the lung or appear in the sputum. The clinical differences between coal-miners' pneumoconiosis and silicosis are chiefly that the coal miner with his focal emphysema and his anthracosilico-tuberculosis is more breathless than ill and his death is like that of a patient with chronic bronchitis, emphysema, and right heart failure. He has miners' dyspnoea or *miners' asthma*. On the

other hand, the patient with silico-tuberculosis is more often ill as well as dyspnoeic. He has *miners' phthisis*, and dies of clinically recognizable tuberculosis.

The clinical course of the illness may cover many years. Sometimes the patient has no symptoms, although routine x-rays show abnormalities in the lungs. And then for a long time the only prominent symptom is dyspnoea. As the disease progresses this gets worse and cough may appear. *Melanoptysis*, the coughing up of coal-black sputum, sometimes occurs and continues on and off for years. The chest becomes barrel-shaped and there may be clubbing of the fingers. Hyper-resonance appears at the bases of the lungs, and here the breath sounds become diminished. When bronchitis supervenes there will be coarse crepitations throughout the lungs. X-rays of the chest show *reticulation* or *pin-head mottling* in both lungs. The progress of the disease is often accompanied by a rising erythrocyte sedimentation-rate. The ultimate clinical picture depends upon whether right-heart failure or silico-tuberculosis supervenes.

In the simple state the dust is found throughout the lungs in the form of black foci measuring up to about 5 mm. in diameter. The dust collects around the small bronchioles and their accompanying arteries, having been brought there from the alveoli by phagocytes. For the most part the dust remains within these cells, the general shape of which is preserved, although the nuclei are obscured. Reticulin fibres develop in the foci of dust. Fibrosis may not proceed beyond this stage, or there may be the development of collagen. The latter does not develop to the same extent as in classical silicosis, nor does it have the concentric disposition, but runs irregularly or radially. The foci have a crenated edge with the processes extending into the unaffected tissues.

In and around the coal foci the air-spaces become dilated, giving a characteristic appearance described as *focal emphysema*. This emphysema has also been described in classical silicosis, but it is very much more severe in the coal worker, and in the latter the emphysematous spaces may enlarge and become confluent. The focal emphysema appears to be due to some mechanical disturbance within the secondary lobules of the lung, as in these units the emphysema starts around the bronchioles and extends

outward towards the interlobular septa. In focal emphysema there are no bullae projecting from the surface of the lung, so that the condition can be distinguished from ordinary bullous emphysema. The development of focal emphysema in coal workers can perhaps be explained by the accumulated dust acting mechanically, the foci of coal dust interfering with the function of the respiratory bronchioles.

Six main methods are used to protect the coal miner against airborne dust.

(i) *Ventilation* is essential in reducing dust concentration. It has been particularly important in the South Wales anthracite mines, where, owing to the methods of working the coal in common use, and owing to the rather haphazard development of the mines from shallow surface workings, poor ventilation was formerly one of the main reasons for the high dust concentrations.

(ii) *Wet Cutting* is achieved by directing two jets of water on the chain of the coal-cutter at the points where it enters and leaves the coal. The usual quantity of water is 5 gallons per yard of advance on the coal face. Of course, this treatment has a very beneficial effect during the subsequent loading on to conveyors. Dry cutting should be forbidden.

(iii) *Wet Drilling* is carried out with compressed-air drills, in which the water passes under pressure through a hollow drill-steel and is directed on to its cutting edge. Dry drilling should be forbidden.

(iv) *Water Infusion* is carried out on coal faces which are not undercut. At intervals of 12 feet along the face a series of 7-foot holes is drilled. A tube with an expansible rubber collar is inserted into each hole and 15 gallons of water at 100 pounds pressure are forced in, a small meter being used to indicate the quantity. The water spreads through the cleavage planes in the coal and wets the loose dust which is always present in the joints and seams. In 1942 this principle was developed in South Wales and it has proved to be one of the most effective methods for dust suppression.

(v) *Hand spraying* is the wetting down with water of the coal face before the coal is pulled down, and of the loose coal before it is loaded, or before it is transferred from one conveyor to another,

or from a conveyor into trams. The doctor must insist that dust-suppression measures should not only be mechanically and physically effective, but that the men who have to operate them should find them convenient and acceptable in use, and that they should by a policy of education come fully to appreciate their need. Sprays that are ill-adjusted may wet the men more than the coal and will be turned off. Excessive use of water in any method may cause unpleasant working conditions. Men are always unwilling to accept measures for their own safety that are inconvenient and require care and attention.

(vi) *Dust Masks.* There are certain mining methods in which the production of heavy dust clouds will be unavoidable, for instance, shot-firing. It will then be necessary for men to wear respirators. Unfortunately, no efficient respirator has yet been devised which men are willing to wear consistently. They cannot do the heavy work of the mine continuously when breathing against the resistance of the filter provided. In the design of respirators, filtration efficiency has been given priority over wearability, despite the obvious fact that an efficient mask in the pocket is of much less use than an inefficient mask on the face. In this field of investigation the biological approach is needed, giving first attention to the convenience of the man, if necessary at the expense of perfect filtration efficiency.

In 1952 the Pneumoconiosis Field Research scheme was undertaken by the National Coal Board, under which all personnel at 25 selected collieries throughout Britain were to be included in regular clinical and radiological examinations and the findings related to working conditions. The existing limits of permissible dustiness could thus be checked and safe standards for air-borne dust concentrations established in British coal mines.

The fight against dust, which includes the employment of fully trained dust-suppression officers in every coal pit, has been going on longest in South Wales. From data available from that coalfield, the potential relation between dust and disease can be noted. In 1943 the rate of compensation for pneumoconiosis was extended. In the South-western Division, which includes the South Wales coalfield, certification rates per 1000 men employed, after rising to a peak of 55·6 in 1945, dropped to 30·8 in 1947. Since

then the falling trend has been maintained and in 1954 the rate was 9·2 per 1000 men employed. As was to be expected, measures for dust suppression have been quicker to show results in the South-western Division than elsewhere. Already working conditions in the South Wales pits have altered out of all knowledge, the levels of air-borne dust in the pits in 1956 being one-tenth part of those prevailing in 1942. Elsewhere in the British Isles it seems that too many cases still go undiagnosed. The rate of certification per 1000 men employed in divisions other than South Wales rose from 0·3 in 1943 to 1·9 in 1947, and the rate for the last complete year, 1954, was 5·8.

Asbestosis

Asbestosis is a special form of pneumoconiosis caused by the inhalation of asbestos dust. Asbestos is a mixture of fibrous silicates, mainly magnesium silicate. Occupations with a possible risk are those involving the working or handling of asbestos, cleaning the carding machines, spinning, weaving, the repair of asbestos textiles, the manufacture of brake linings, and the lagging of boilers and hot-water pipes.

The lesion in the lungs is not in the shape of discrete nodules but instead there is diffuse fibrous scarring accompanied by pleural thickening and emphysema. The inhaled asbestos fibres undergo a change in the lungs and become converted by the deposition of fibrin into the characteristic *asbestosis bodies*. Under the microscope these are seen to be club-shaped segmented rods up to 200 microns in length. They are formed around asbestos fibres.

The symptoms are dyspnoea, cough, and expectoration. The physical signs are cyanosis, clubbing of the fingers, and fine crepitations at the bases of both lungs. The sputum contains *asbestosis bodies* which may be regarded as of diagnostic significance only if they are present in clumps. Radiographic changes are late in developing, and the symptoms and signs may be present before they appear. The first x-ray findings are a ground-glass appearance or a very fine stippling of the bases of the lungs; the heart shadow may present a shaggy outline and the diaphragmatic border is obscured.

Preventive treatment consists of rigorous dust suppression. In asbestos mining, which is carried out on a large scale in Canada, wet rock drilling is imperative. In Great Britain in factories and textile mills the principle of locally applied exhaust ventilation is enforced to prevent the escape of asbestos dust into the air of any room in which work is done. In the manufacture of asbestos textiles, hand cleaning of the cylinders of carding machines is prohibited while any other person is present, nor may it be done by hand strickles or other hand tools. Those cleaning cylinders, filling insulating mattresses, and entering chambers containing loose asbestos, should be provided with an efficient apparatus by means of which they can breathe air free from dust (Plate 12). Suppression of dust in the cleaning of carding machines is best ensured by the use of a revolving brush fitted with a cover and connected to a portable vacuum cleaner. By the use of closed-in machines, handling should be reduced to a minimum. In one factory, spinning frames have been totally enclosed in *Perspex* with an ingenious device which permits easy access to bobbins. Education of the worker must aim at a high standard of good housekeeping. Dr E. R. A. Merewether, late H.M. Senior Medical Inspector of Factories to the Ministry of Labour and National Service, made extensive investigations of the dust hazard in this industry and upon this work the present methods of protection are based.

Byssinosis

Byssinosis occurs in workers in the cotton industry employed for many years in cotton rooms, blowing rooms, or card rooms where the spinning of raw cotton is carried on. The workers are exposed to the inhalation of dust arising in these processes. The course of the disease suggests that it may be the result of the type of sensitivity known as allergy.

The established condition is one of chronic bronchitis and emphysema. The symptoms are progressive dyspnoea, sometimes asthmatic in type, with cough and, at first, scanty sputum. The early stages are often called *Monday fever* because there is a difficulty in breathing on resuming work on Monday mornings or after an interval away from work. At first this quickly passes,

but after some years the respiratory disability becomes continuous. It increases until in about 20 years complete incapacity may result. The radiograph shows no characteristic change but is useful for eliminating other lung diseases. Right heart failure is the usual cause of death. The disease is very slow in onset and may cause no disability for 10 years or more (see page 79).

Attempts at *preventive treatment* by dust suppression in cotton mills began in 1908. Obviously the ideal method is to ensure that the cotton dust does not escape into the atmosphere. In 50 years this has not been found possible. Considerable improvements have been achieved by the introduction of dust covers and exhaust fans for machines in the cotton chamber and blowing rooms. Unfortunately, even in the best-equipped mills, at least one-third of the trash is carried into the card room in the laps; it is this third which is eliminated in the carding process. The sliver emerging from the carding engine is almost dust-free.

The under-space of the carding engine should be completely sealed off, and to clean under the engine the stripper and grinder has only to pull a small lever; the entire collection of dust is then swept away by the creation of a vacuum. This arrangement has greatly reduced the amount of dust in the atmosphere. In some mills one per cent mineral oil is sprayed on the cotton in the hopper bale-breaker. This treatment helps to reduce the dust and does not interfere with the quality of the yarn since most of the oil is absorbed before this stage is reached. Many mills have been built with insufficient air space and therefore the machinery is crowded into too small an area. Greater air space means a greater dilution of the dust. Improved air conditioning, in conjunction with improved dust extraction, is necessary. Air conditioning must include a correctly controlled temperature, the right humidity, and adequate air movement. These are also the best conditions for the cotton fibre.

The engineering problems involved are complex and the British Cotton Industry Research Association at the Shirley Institute, Didsbury, Lancashire, is continuously employed in researches on the subject. A new report entitled *Dust In Card Rooms: Third Interim Report of the Joint Advisory Committee of the Cotton Industry, 1957*, recommends the Shirley pressure-point system

of exhaust ventilation in card rooms. The three cylinders of the carding-engine act as a fan, creating zones of high and low air-pressure. The Shirley system is designed to relieve the high-pressure zones and remove the dust which would otherwise be liberated from them into the atmosphere. The device has been exhaustively tested at the Shirley Institute and in a full-scale installation in a mill. In a foreword to the report, Sir George Barnett, H.M. Chief Inspector of Factories, commends this system as a means of preventing byssinosis.

A careful selection of operatives for the card room, and regular medical examination of existing workers, offer the best protection. All intending card-room operatives should be medically examined before engagement, and every effort should be made to exclude those likely to be susceptible to the disease. Any operative who gives a personal or a family history of asthma, bronchitis, hay fever, eczema, or recurrent urticarial attacks should be excluded from the card room. The general build and shape of the thoracic cage should be noted, and also the type of respiration. Mouth breathers are bad subjects. Malformation of the nasal bones, deflected septum, nasal polypi, enlarged tonsils, and extensive adenoid vegetations are all contraindications for work in the card room. A chest expansion below 3 inches is suspect. All operatives are urged to practise nasal inspiration and to wear protective respirators.

In spite of all the improvements introduced, a substantial number of men employed in the more dusty mills spinning the coarser grades of cotton still suffer from byssinosis. There could be a more effective control of the disease if medical supervision and engineering control of dust were combined. There is at present no system of periodical medical examination of card-room workers. If there were, the incidence and severity of the disease found would indicate both the mills where improved methods of dust control were needed, and the men who were getting progressively worse and who should be encouraged to leave the industry or work in the less dusty mills. Dr Richard S. F. Schilling of the London School of Hygiene and Tropical Medicine, London, has made noted contributions to this subject.

CHAPTER EIGHT

OTHER OCCUPATIONAL DISEASES

Occupational Diseases Due to Infections – Anthrax – Glanders – Weil's Disease – Compressed-air Illness – Occupational Cramps – Writers' Cramp – Telegraphists' Cramp – Cotton-twisters' Cramp – Miners' Nystagmus – Injuries from X-rays – Injuries from Radioactive Substances – Injuries from Fission Products – Occupational Bursitis – The Beat Disorders of Miners – Occupational Diseases of the Skin – Occupational Cancer

IN this chapter is considered an assortment of disabilities due to infections and to physical causes such as compressed air, x-rays, and radioactive substances. Happily the number of such cases is small. The same cannot be said of occupational dermatitis and of occupational cancer, where unhappily the cases which still occur are far too numerous.

OCCUPATIONAL DISEASES DUE TO INFECTIONS

Conditions peculiar to certain trades may cause disease which predisposes to infections. Thus silicosis leads to an excessive mortality from pulmonary tuberculosis. There is likewise a heavy mortality from tuberculosis in publicans, cellarmen, barmen, wine waiters, brewers' draymen, and others who have ready access to alcohol. The effects of dissipation, however, are marked in only a few occupations, and other factors, such as overcrowding, bad ventilation and lighting, cramped and sedentary occupations, over-fatigue, and excessive hours of labour, play their part in spreading tuberculosis.

Workers exposed to great heat such as occurs in the steel and glass industries show a greater liability to pneumonia than other workers. Persons exposed to organic dusts also seem to have a susceptibility to pneumonia. The dusts of bagasse, bone, cotton, derris, feathers, flax, flour, fur, grain, gum arabic, hair, hay, hemp, horn, ivory, jute, leather, linseed, malt, nuts, paprika,

seeds, silk, sisal, straw, tea, tobacco, wood, and wool may attack the bronchi and thus give rise to an increased incidence of respiratory diseases among the men who work in them. Silicosis is productive of death from pneumonia to a degree only secondary to its influence in predisposing to phthisis. Pneumonitis may be determined by the inhalation of chemical substances, especially compounds of beryllium, manganese, osmium, and vanadium.

In certain occupations workmen are exposed to the risk of infected wounds. The immediate aseptic or antiseptic treatment of all cuts and abrasions will go far to prevent these infections, and such measures are of great value in reducing disablement in industries where minor injuries are of frequent occurrence, such as the making of barbed wire.

Animal infections may be transmitted to man in the course of his work. Anthrax may occur in men handling carcases, skins, hides, or hair of animals that have been infected. Glanders is occasionally derived from contact with sick horses. Jute has been the means of conveying tetanus, owing to the large number of spores in the soil and dirt with which jute is always mixed. Ectothrix organisms are known to affect horses, cattle, cats, and birds: four clinical types of ringworm are recognized in man from contact with these creatures. Brucella infections may occur in men attending cattle and pigs, or handling beef and pork in abattoirs. Parrots, budgerigars, and fulmar petrels suffering from psittacosis may convey infection to those whose work involves handling them, and extensive outbreaks of the disease are now well known. Gardeners, veterinary surgeons, housewives, and research workers may be infected.

The cowman and unvaccinated nursemaid occasionally suffer from vaccinia, the former infected from the udders of a cow and the latter from direct contact with a vaccinated baby. Doctors and nurses are liable to accidental infection from patients suffering from typhoid, diphtheria, gonorrhoeal ophthalmia, and streptococcal sore throat, as also to septicaemia from puncture wounds and to primary chancre of the finger. In certain circumstances they may become infected by poliomyelitis, anthrax, typhus, cholera, or plague.

ANTHRAX

Anthrax is a disease which may occur both in man and in certain animals as a result of infection with *Bacillus anthracis*. Cattle are the main source of infection. Man may become infected through contact either with material containing the bacillus, as in agricultural workers, veterinary surgeons, butchers, and knackers; or with dried animal products such as hides, skins, hair, wool, horns, hooves, and bone-meal harbouring the spores, as in dock-workers, tannery and wool workers, and workers in factories where bones are crushed to make fertilizers. Hides, skins, and hair from the Far East, and from Siberia and parts of Africa are liable to be infected, as are also wool and hair including alpaca, mohair, goat, and camel hair from the Middle East.

Infection in man may be cutaneous, *malignant pustule*, or pulmonary, *wool-sorters' disease*. Gastro-intestinal infection is rare. The disease is of sudden onset with grave toxaemia, headache, shivering, pains in the limbs, nausea, vomiting, and collapse, together with local symptoms depending on the site and type of infection.

Malignant pustule is the commoner form. Infection takes place through the skin of some uncovered part of the body and leads after 1 to 4 days to a pink itching papule which enlarges and develops a black centre ringed round with small vesicles (Plate 13). Associated lymph glands become enlarged and tender. In the case of the face and neck intense oedema surrounding the lesion may itself be a danger to life. The diagnosis is rendered certain by discovery of the bacillus in the discharge, or by its recovery after animal inoculation. In most cases recovery takes place by separation of the scab and healing of the wound. The use of penicillin has reduced the death rate virtually to nothing.

Pulmonary anthrax produces intense toxaemia, with sudden vertigo, somnolence, dyspnoea, and marked prostration. Death may supervene from bacteriaemia without the appearance of localizing symptoms and signs. Sometimes broncho-pneumonia with frothy blood-stained sputum occurs. Provided the diagnosis

is made in time penicillin saves life even where anthrax bacilli can be recovered from the cerebro-spinal fluid.

The *preventive treatment* of anthrax is of supreme importance. Since it is difficult to prevent animals all over the world suffering from anthrax, it is necessary for Great Britain to disinfect infected material which she imports. Disinfection varies according to the material under consideration. For horsehair, steam disinfection is practicable if the temperature inside the autoclave does not exceed 230° F. The fibres of wool, however, would lose their elasticity and be destroyed for manufacturing purposes by this method. The *Anthrax Prevention Act, 1919*, regulates the importation into the United Kingdom of certain goods likely to be infected with anthrax, and orders made under it apply to goat-hair from any part of the world and to wool and hair coming from Egypt, which it requires to be landed and disinfected at the port of Liverpool.

There is need for close supervision and attention to detail in the disinfection of wool, since this can be made completely safe. Unhappily no satisfactory process has been evolved for the treatment of hides and skins without causing damage to the material. The Government has erected a Disinfecting Station in Liverpool to treat the most dangerous kinds of wool and hair. The process used was elaborated by Mr G. E. Duckering, one of H.M. Engineering Inspectors of Factories, and by Dr F. W. Eurich, and is known as the *Duckering process*. It kills all anthrax in any sample of wool or hair without detriment to the material or harm to the workers. The bales are placed on a platform and the wool or hair is carried automatically into a bale opener which is enclosed and provided with a dust-extracting apparatus. The wool is then moved slowly through a succession of large troughs by stainless-steel prongs attached to a harrow-like frame.

Each batch of hair is treated for ten minutes in each of five troughs. The first contains a 0·5 per cent solution of sodium carbonate in water, and the second a 0·5 per cent solution of soap in water containing a little free sodium hydroxide. Both solutions are kept at a temperature between 102° and 110° F. The material is constantly agitated in the solutions by means of rakes, and between each trough passes through heavy squeeze rollers. This

preliminary treatment brings the anthrax spores into a condition in which they are easily killed. The third and fourth troughs both contain a 2 per cent solution of formaldehyde in water at a temperature of from 102° to 105° F. Finally the material is rinsed in clean water at the same temperature to remove excess formaldehyde, passed again through squeeze rollers, and dried in a current of air at a temperature of 160° F. It is then press-packed into bales of 375 pounds.

Control of the strength of the solutions and of the temperature is essential to the success of the process, which aims at achieving complete disinfection without damaging the materials. Routine chemical examination of the solutions and bacteriological examination of materials before and after disinfection ensure that the process is efficient. It is claimed that, in twenty-five years, there has not been a single case of a sample still containing anthrax after being subjected to the *Duckering process.*

In factories and workshops where there is a risk of anthrax, dust must be removed by downward exhaust ventilation. It is impossible to remove such dust completely, but in spite of this, cases of internal anthrax are now very rare. Many shipping firms have provided protective gloves for their workmen, but the difficulty of getting the men to wear them is great, and there is some risk that they may harbour infection by dust finding its way into the inside of the glove.

One vital aspect of prevention is the warning given to working people of the importance of early recognition and prompt treatment. To attain this end, *cautionary placards* have to be displayed in certain factories by Regulations. These show coloured pictures of the skin lesion in anthrax at various stages. Such notices draw attention to the means by which the disease is communicated, the need for its prompt recognition, and for immediate attendance at a hospital. Not only has the worker been warned, but it has been thought advisable to issue a caution to medical practitioners. Anthrax is not a common disease, and it is not surprising that it is overlooked in its early stages. To assist doctors and to ensure the prompt treatment of anthrax, an *individual card* has been devised by the Factory Department of the Ministry of Labour for workers employed in industries exposing them to risk. Such

individual cards when presented to the doctor suggest tactfully to him the possibility of anthrax infection and the need for a more extensive examination in the case of a worker who presents such a card.

Glanders

Glanders is now rare in Great Britain. It is a communicable disease caused by infection with *Pfeifferella mallei*, which affects chiefly horses. Although transmissible directly from man to man, infection usually comes about from direct contact with an infected animal. The bacillus gains entry through skin wounds or bites or through the undamaged mucous membrane of the eye, nose, mouth, or gastro-intestinal tract. The disease is a granulomatous infection and except in length and severity there is no difference between the acute and chronic forms. In some cases the lesions are limited to the skin and the condition is then called *farcy*.

In the *acute form* after an incubation period of from 2 to 3 days the illness begins with general malaise, headache, anorexia, nausea, and joint pains followed by swelling and ulceration at the site of infection, with marked lymphangitis and painful local adenitis. Pyaemia supervenes with enlargement of the liver and spleen and the appearance of tumours in the skin which quickly suppurate and discharge, leaving painful ulcers. At this stage there is high fever, and a characteristic eruption appears affecting the face and the mucous membranes of the eyes, nose, and mouth. The lesions begin as pink macules and rapidly become papular and then pustular, particularly in the nose. Here painful ulceration and destruction of cartilage and bone occur, accompanied by a bloodstained, mucopurulent discharge. Death usually supervenes in 3 weeks or less.

In the *chronic form* glanders is a long painful illness lasting many years. The granulomatous lesions suppurate and produce painful sinuses. The illness may become acute at any time, but death usually occurs from an intercurrent infection.

Diagnosis of human glanders is difficult. A history of contact with horses may indicate the nature of the illness. In acute cases

the early symptoms may suggest pneumonia, typhoid fever, or rheumatic fever. *P. mallei* can be demonstrated easily in the lesions of acute glanders and during exacerbations in chronic glanders, but when the lesions in the chronic disease are more or less stationary it may be extremely difficult to confirm the diagnosis. Cultures should be made on glycerine agar and potato, and pus from the lesions inoculated into male guinea-pigs intraperitoneally. Complement fixation is the most reliable laboratory test.

In *treatment* large doses of sulphadiazine given for 20 days may be effective. Little is known of the effectiveness of the modern antibiotics, but the organisms should be tested for sensitivity, and the appropriate substance used. Treatment includes conservative surgical drainage of abscesses.

Weil's Disease

This disease, known also as leptospiral jaundice, spirochaetal jaundice, and spirochaetosis icterohaemorrhagica, is an infection caused by the *Leptospira icterohaemorrhagiae*. Infection may be due to ingestion of food or water contaminated by the urine of infected rats; or the organism may enter through the skin or through the mucous membranes of the eyes, nose, or mouth. The disease sometimes occurs among men who work in places that are rat-infested, as for example sewermen, miners, bargemen, wharfmen, fish and offal workers, and workers in slaughterhouses and piggeries.

After an incubation period of from 6 to 12 days there is an abrupt onset of high fever, rigors, headache, muscular pains, and vomiting. The face is flushed, there is intense injection of the conjunctivae, and prostration is marked. The fever goes on for 3 or 4 days, and then subsides by slow lysis, but the temperature often rises again towards the end of the second week, and persists for another week. In 60 per cent of cases jaundice appears on the second or third day of the illness. In such cases the liver is tender and the spleen may be enlarged. At this stage herpes febrilis at the angles of the mouth is common, and a papular or, less often, a petechial rash may appear on the skin.

The urine is scanty, high-coloured, often bile-stained, and may show albumin. There is a tendency to sleeplessness, with nocturnal delirium, and this may be accompanied by meningeal symptoms. Haemorrhages, and especially epistaxis, are common.

The *diagnosis* is usually made in the laboratory, the leptospira being recovered from the blood during the first week, or from the urine up to 6 weeks from the tenth day. Serological tests show the presence of agglutinins in dilutions upwards of 1 in 100 after the eighth day, and the titre may rise as high as 1 in 30,000. A rising titre is, of course, diagnostic. At the height of the illness there is usually a polymorphonuclear leucocytosis.

In the *preventive treatment* of Weil's disease intensive and systematic destruction of rats should be carried out in infested mines, sewers, docks, ricefields, and sugar-cane fields. Much control of the disease could be achieved by cleanliness campaigns for removing material soiled by the urine of rats, such as fish offal. The disease is always found to be associated with wet conditions. In mines it should be controlled by pumping dry those sections which are wet. In Japan *L. icterohaemorrhagiae* thrive in the water of the paddy-fields, and coolies working in these fields often suffer from Weil's disease. It has been found that when calcium cyanamide, which readily kills the organism, is used as a fertilizer, none of the coolies become infected. Control of the disease in the case of sewer workers presents difficulties, and prophylactic immunization would appear to be the best means available. Each worker should carry an individual card which teaches him to wash his hands before meals and serves to warn the doctor of the possibility of infection. No man should be employed unless he shows a positive reaction to an agglutination test for leptospiral antibodies. A doctor should visit each sewerman who is off sick so that a diagnosis can be made as early as possible in the disease.

In *curative treatment* both antiserum and penicillin must be used as early as possible. Penicillin has a bactericidal and bacteriostatic action on *L. icterohaemorrhagiae*. It must be administered within the first 4 days of the onset of septicaemia, otherwise its action can avail the patient little, especially if nephritis develops.

Compressed-air Illness

Work in compressed air has to be done in civil engineering involving excavations in water-bearing strata or under water, as well as in diving. Such work is effected either by men in diving suits, or by a group of workers in a caisson or diving bell. A caisson consists essentially of a working chamber and a shaft communicating at the surface with an air-lock which in turn communicates with the outer air. All these receive cooled compressed air from a pipe-line. Within the caisson proper, in which the men work, the air pressure must be equal to the pressure exercised by the water outside, and must therefore be raised in proportion to the depth at which work is being carried out. It is raised approximately 1 atmosphere for each 10 metres of depth. In practice, the pressures used do not usually exceed 3½ atmospheres, that is, about 40 pounds per square inch.

While men are being subjected to increased air pressure in the air-lock preparatory to descending into the caisson, discomfort may occur in the ears from disparity of air pressure in the middle ear, *ear block*. If air under pressure is trapped in the nasal sinuses, there may be pain, *sinus block*. Abdominal pain and distension may occur from trapped gases in the intestinal tract. It is not until later, during decompression and return to lower pressures, that the symptoms of *compressed-air illness*, or *caisson disease*, are liable to appear. The symptoms of this condition are due to the release within the blood and tissues, when the air pressure is reduced, of gases previously driven into solution when the air pressure was high. In fact the gases within the body of a patient too rapidly decompressed behave like the carbon dioxide inside a soda-water siphon when the pressure is reduced by depressing the trigger. In the patient the gases concerned are oxygen, carbon dioxide, and nitrogen; the two former are removed very rapidly, the first by reabsorption and the second by exhalation from the lungs, but nitrogen is relatively insoluble in the body fluids and tends to collect in the tissues as minute bubbles of gas. Since nitrogen is 5 or 6 times as soluble in fats and related substances as in the body fluids, tissues such as

the body fat, the nervous system, and bone marrow hold proportionately more of the gas than others, releasing it in bulk when the pressure is reduced.

Symptoms usually appear within the first few hours following decompression, but may not develop for 12 hours or more. They depend on the location of any nitrogen bubbles formed. The commonest sympton is severe pain in the muscles of the limbs, and it is this condition, due to the presence of nitrogen bubbles in the muscles, which is called *the bends*. Erythema of the skin with pruritus, *the itch* or *prickles*, occurs with regularity if the skin is chilled during decompression. Vertigo, *the staggers*, is a common symptom. The staggering gait may be accompanied by nausea, vomiting, tinnitus, and nystagmus. A type of asphyxia known as *the chokes* may occur, though it is rare. In some cases there is involvement of the central nervous system, and symptoms and signs such as paralysis of skeletal muscles or of the bladder may appear. Paraplegia was at one time so common in divers that it became known as *divers' palsy*. Nitrogen emboli in the blood vessels of the lungs, brain, or heart may be fatal. *Aseptic necrosis* of bone may occur especially in the femur and humerus, either in the diaphysis or epiphysis. As elsewhere, such lesions are the result of infarction. Bone and joint lesions may not be noticed for some time after the incident, and by then there may be some attempts at repair, but areas of dead bone, sometimes involving damage to articular surfaces, can often be demonstrated by x-rays long afterwards. Such lesions may result from a single over-rapid decompression.

The *preventive treatment* of compressed-air illness depends upon adopting suitable limitation of exposure. The malady never arises from compression below 18 pounds to the square inch, or roughly 40 feet of water, and those who work at such a pressure may do so for long hours and return to a normal pressure rapidly and without any risk. At higher pressures the working shifts must be shortened as the pressure gets higher. The shifts should not be longer than 6 to 8 hours at a pressure of from 30 to 35 pounds per square inch, or 3 atmospheres; 2 to 3 hours at a pressure of 45 pounds per square inch, and 1 hour only at a pressure of 50 pounds per square inch. At higher pressures than this, which

are encountered only by divers, only a few minutes' exposure is allowed. The regulation must be strictly enforced that no person employed shall consume alcohol whilst in compressed air.

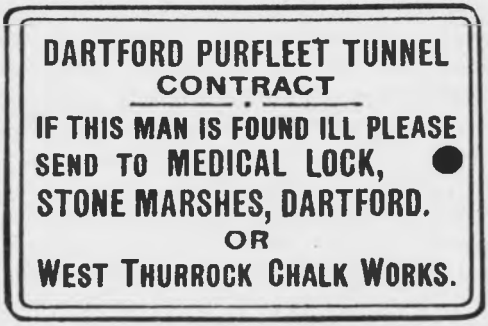
Compressed-air illness never occurs if the return to the normal atmospheric pressure be sufficiently slow. In the case of caisson workers a series of air-locked chambers is provided in which the air pressure is lowered in stages, the men remaining longer and longer at each stage as they approach the normal pressure. The important fact in connexion with decompression is that the absolute pressure can always be halved forthwith without any risk. In the first air-lock on leaving the working face of a caisson, for example, the pressure is at once reduced to one-half that of the working-face, and in the remaining air-locks the pressure is reduced by stages until zero is reached. The difficulty and danger is the tendency on the part of the workers to curtail these weary waits in order to get away from work as soon as possible. The supervision of men working in caissons must be strict and continuous. Even when careful precautions are devised they do not wholly eliminate the risk of serious illness, for a man may let himself out through the air-lock normally used for the disposal of rubble from the working site and develop paraplegia in consequence.

In the case of the diver decompression is carried out by raising him to various levels in stages, and letting him remain at each stage a longer and longer period as the surface is approached. The Admiralty rules for divers require that a diver working, say, at 140 feet shall be first raised straightway to a depth of 50 feet where he waits 10 minutes, then to 40 feet for 10 minutes, 30 feet for 20 minutes, 20 feet for 30 minutes, 10 feet for 35 minutes, and then he leaves the water abruptly.

It was early discovered by the caisson workers themselves that the only remedy for the malady was to re-enter the high air pressure. A recompression apparatus in the form of a medical air-lock is supplied at all caisson works and in all ships engaged in deep salvage. On the appearance of any symptoms the worker is placed in the compressing room and the pressure is run up to the level at which he has been working, when it is usual for the symptoms to diminish rapidly or to disappear.

After the recompression the decompression must be carried out very slowly, for the nitrogen bubbles once formed in the tissues are not easy to get rid of, though they may be kept at a small size by the pressure. A patient apparently at the point of death with cyanosis and coma may completely recover in a few hours by recompression. When symptoms have appeared, the decompression should take from five to twenty-four hours.

Caisson workers and divers should wear on the lapels of their jackets a metal label clearly inscribed with a notice stating both their occupation and the address of their place of work.



They should sleep and live close to the medical air-lock in order that they may be near aid during the first hours following decompression. The paralysis usually needs to be treated by rest in

bed and proper nursing care. Aseptic necrosis of articular cartilage may necessitate opening a joint for removal of necrotic material. Supervision of the health of workers in compressed air, whether they are employed by commercial organizations or by government departments, has been brought up to date in the *Work in Compressed Air Special Regulations, 1957.*

Occupational Cramps

The term occupational cramps is used for a group of conditions in which certain symptoms are excited by the attempt to perform a customary act involving an often-repeated muscular action. The necessary co-ordination of movement breaks down, and spasm, tremor, pain, weakness, and loss of control occur in the muscles accustomed to perform harmoniously the regular act concerned. These conditions are also known as craft palsies, occupational palsies, and professional spasms.

The hands are affected in writers, telegraphists, cotton twisters, tailors, drapers, seamstresses, sailmakers, knitters, hairdressers, ironers, metal workers, hammermen, turners, engravers, goldbeaters, cabinet makers, sawyers, locksmiths, tinsmiths, nailmakers, masons, painters, enamellers, compositors, watchmakers, shoemakers, saddlers, sailors, fencers, diamond cutters, money counters, letter sorters, cigarette rollers, cigar makers, pianists, organists, violinists, violoncellists, harpists, flautists, drummers, orchestra conductors, typists, comptometer workers, waiters, florists, artificial-flower makers, folders of newspapers, and milkers of cows. Rarely the lower limbs are affected, as in dancers, sewing-machine workers, knife sharpeners, and tradesmen's tricyclists. Identical spasms of the muscles of the head and neck are still rarer. Cramp of the tongue in clarinet players and of the lips in trumpet players have been described.

Most of these occupations involve rapid, repetitive movements of short range, either by one or by both hands. The movements concerned are complex and are perfected by education and practice. They necessitate a high degree of precision and co-ordination and they may involve as many as 10 repetitive movements a second. The first manifestations of the disorder are likely to

make their appearance when the worker is called upon to exceed a certain level of performance. There may therefore be an associated anxiety on the part of the individual to get the work done in time and up to standard. Symptoms may appear after physical or psychological events which lower the patient's normal level of efficiency.

No structural change in the cerebral cortex, nervous system, or muscles has ever been demonstrated. By careful and detailed clinical interview it is possible in many cases to elicit the presence of psychoneurotic symptoms. Opinion has gradually moved away from the conception of the disorder as being due to physical fatigue of muscle or nerve towards the view that it is primarily a disorder of behaviour. The causative factors are no doubt multiple and both physical and psychological in nature. They result in the breakdown of the smooth execution of a stereotyped movement and ultimately lead to the setting up of a faulty habit.

Writers' Cramp

Writers' cramp has also been called writing neurosis, scriveners' palsy, and graphospasm. Most commonly the presenting symptom is motor spasm, but besides the spastic type there are also tremulous and neuralgic types of the disease. The paralytic type with simple inability to write is extremely rare. The onset is gradual. After writing for some time the patient finds that the pen does not move quite as he intended it to do; a stroke now and again is irregular, extends too high or too low; a slight involuntary movement causes an unintended mark. He finds that he is grasping the pen too tightly, and cannot help doing so; that the fingers do not keep in their accustomed place; and the first finger has a tendency to slip off the pen, so that this gets between the first and second finger. Taking a firmer hold seems only to increase the difficulty, and he finds that he writes slowly, as if a weight were attached to the hand. The hand feels tired, and an aching pain in the finger, thumb, wrist, or forearm makes it still more difficult for him to go on writing. These symptoms may continue, with only slight impairment of the power of writing,

for weeks or months, but they occur after writing for a shorter time; they increase in degree, and now and then there is distinct spasm, which cannot be controlled. The first finger or the thumb tends to become flexed at the middle joint, so that its tip moves up the pen, or, less commonly, the fingers become extended, so that the pen is not pressed against the paper with sufficient force, and may even drop from the hand, or the thumb may become extended across the pen. The characters of the writing become still more irregular, the point of the pen may be driven through the paper, and in its irregularity of form and force the writing resembles that done in a jolting vehicle.

Power in the hand may be slightly impaired; wasting is exceptional. The electrical reactions are only slightly disturbed, if at all. Sensory symptoms are usually prominent and rarely absent. A sense of fatigue in the hand is followed by spasm and dull pain, occasionally with local tenderness. The pain may be referred along the course of nerves; paraesthesiae may affect the fingers. The symptoms continue with the attempts to write. The sensory symptoms spread more than the motor. In about 50 per cent of those who have learnt to write with the other hand, symptoms appear for a second time and may progress more rapidly in the second arm than in the first. No anatomical changes are known. Clearly the condition is one of central and not of local origin, since local weakness is only found to a degree comparable with spasm. The theory of central origin is supported by involvement of the opposite hand. That the pain starts from the skin where the grasp of the pen is unduly firm, but spreads beyond this point, is evidence in favour of a central origin. The majority of patients are nervous subjects; in showing the signs and symptoms of pathological anxiety they reveal the psychoneurotic personality.

In *treatment* writing should be taught with free movements from the shoulder. It is also desirable that those who experience any difficulty or discomfort in writing should at once change their style for the freer mode. Let the patient first draw a line across a sheet of paper, moving the arm as a whole from the shoulder. Then let him make the line wavy, next increase the wavy character, and then slightly slope the waves, so as at last to make the

line a series of m's – m m m m. The transition to other letters will then be easy. The object to be aimed at is to write in such a manner that it shall be easy to form an entire line of words without once lifting the pen from the paper. The hand should grasp the pen lightly, and move as a whole. Considerable comfort and ease are experienced when this method is acquired. Treatment, to be effective, must be early. Rest may relieve the symptoms within a month, but in well-established cases six months will be required. Facility in writing with the unaffected hand is not difficult to acquire. A typewriter may be recommended for recalcitrant cases. Where there is pain, sedatives are certainly of service. Electricity is of little use, but massage and exercises often relieve the spasm and pain.

Telegraphists' Cramp

This disability occurs in telegraphists using the Morse key for prolonged periods. The Morse key is a clumsy brass instrument, the knob of which is depressed by hand in a series of rapid movements which make and break the circuit in such a way as to transmit the dots and dashes of the Morse code. Telegraphists' cramp is a spasm of the muscles of the hand and arm, with some incoordination of movement. It is the result of prolonged muscular fatigue in a person of nervous temperament. The disability tends to develop in skilled operators rather than in learners. The patient complains of spasm of the muscles of the hand, with tremor and muscular weakness. Particular combinations of dots and dashes prove stumbling blocks. Some have difficulty in letters involving a sequence of dots, particularly at the end of a word; others find that having got the key down they cannot get it up again quickly enough for the formation of dots. There are no signs of muscular wasting or of sensory loss. The muscles concerned respond normally to electrical stimulation.

As to *treatment*, mild cases recover when prolonged rest is ordered. In many of the severe cases where the holding of a cup or the use of a pen is difficult or impossible nothing short of an entire change of employment will cure the condition. The disappearance of anxiety naturally has a good influence on the

prognosis. The problem has been solved by mechanization in the form of the tape machine. Automatic recording systems which are essentially letter-printing instruments have now come into extensive use. The teleprinter is an electrically operated typewriter with a standard keyboard. It has replaced the Morse key almost universally. The extended use of the telephone also has helped the situation. In places where the pressure of telegraphic work is slight only, as in isolated country post-offices, the messages are relayed by telephone to the nearest telegraph station.

Cotton-twisters' Cramp

In the Lancashire cotton trade there is a form of occupational cramp which may suddenly attack twisters of many years' experience. In joining the threads of a warp, the weaving of which has been completed, to the threads of a new warp the two ends are twisted between the flexed left thumb and the dorsum of the left index finger. An experienced man may join 2000 ends in one hour, each warp having between 2000 and 2500 threads. The symptoms are pain, cramp, and loss of power in the affected muscles, and about 50 per cent of the patients show wasting of the thenar eminence. It is a disability causing much loss of earning power in the men affected, and anxiety makes it worse. Treatment by massage and exercises, though alleviating the condition, will not permanently cure it. In severe cases an entire change of employment will be found necessary.

Miners' Nystagmus

The disease known as *miners' nystagmus* is the result of poor lighting in coal pits. Because the presence of methane acted as a brake to efficient lighting there was a time when large numbers of coal miners had worked in the dim light of flame safety-lamps over a number of years. Miners' nystagmus is confined to this group of men. In Great Britain measurements have shown that the illumination at the coal face in naked-light pits is generally from 5 to 10 times as great as that in safety-lamp pits. Where

there is no methane hazard as in naked-light coal mines, metalliferous mines, and slate mines, the men are found to be practically free from the disease. In the United States of America coal-face lighting has always been vastly superior to that in Europe. They passed from the naked acetylene flame to the electric cap lamp giving a light from 20 to 50 times brighter than that of the flame safety-lamp. There is no good evidence to suggest that miners' nystagmus is caused by the awkward posture necessitated in coal mining, variations in the temperature and humidity of the pits, infection, or inhalation of carbon monoxide, methane, or nitrogen dioxide.

The disability may be discovered accidentally in a symptomless form, or it may be brought to notice, usually in a person of nervous temperament, as an incapacitating disease. In the latter case some precipitating factor such as the shock of an accident or the onset of an acute anxiety state is usually discernible. The symptom of oscillation of lights is sometimes present. But oscillation of the eyeballs with a rapid rotatory rhythm may be symptomless even when well marked. In such cases the miner has acquired a tolerance to the oscillation; his cerebral interpretation of the stimuli from his roving eyes is that of a normal image. Where incapacity accompanies nystagmus the symptoms usually include headache, vertigo, and insomnia. In such cases there may be some contraction of the fields of vision, poor visual acuity, and photophobia, these disabilities being often associated with nervous symptoms and signs such as tremor of the head and stiff neck. There is usually at some stage oscillation of the eyeballs, sometimes with fluttering of the eyelids, but these signs may be intermittent, may not be evident at examination, and may disappear altogether before the other symptoms yield to treatment.

Treatment must be considered carefully for each case. Thus a man with nystagmus and marked symptoms should be considered as unfit for work underground. Many such men can perform certain forms of surface work, but work involving stooping should not be included for fear of aggravating the oscillations and the vertigo. Work on the screens is thus not suitable, or work involving heavy digging or the filling of trucks or wagons. If vertigo is

severe, work amongst moving machinery or traffic also should be excluded. Incredible though it may sound, it is the custom of some medical men to tell their patients that they must never go down the pit again unless they wish to become blind. No man loses his sight from miners' nystagmus, and the first duty of the doctor is to assure the patient that his condition is not incurable and to re-establish the man's confidence in himself by stating most pointedly that blindness never results.

The reflecting power of coal is small, ranging from about 5 per cent in the case of dull coal to 10 per cent in the case of bright coal. Shale and other rocks met with in coal mines reflect from 20 to 30 per cent, whilst supports and props usually reflect from 10 to 20 per cent. It will be obvious that the lighter the surfaces the more light is usefully employed. Whitewashed surfaces reflect anything from 50 to 60 per cent of the light falling upon them. The *Coal Mines (Lighting) General Regulations, 1947*, enumerate places which are to be whitened:

(*a*) such of the shaft insets and sidings as are regularly used;
(*b*) the top and bottom of every permanent self-acting incline;
(*c*) every siding, landing, passbye, junction, offtake, place at which tubs are regularly coupled or uncoupled or regularly attached to or detached from a haulage rope, and place at which tubs are regularly filled mechanically, except in so far as any such place as aforesaid is within 100 yards of the face; and
(*d*) every room and place made to house, and containing, any engine, motor, electrical transformer, or switchgear.

The provision of better lighting in coal mines is *preventive treatment* which has done much towards the eradication of this disease, reducing the number of new cases and alleviating the misery of existing sufferers. Thus in England the number of men certified as suffering from miners' nystagmus reached its peak in 1922, when 4092 cases were recorded – that is, 1 out of every 225 miners employed underground. By 1938 the number of cases had fallen to 1224 or 1 in every 510 employed underground. For various reasons the number rose again during the Second World War to 2006 in 1943 or about 1 in every 300, but it has since fallen to 641 cases in 1948 or 1 in 830 employed underground.

These figures show that progress is being made, and there is hope that with improvement in the quantity and quality of underground lighting we shall see nystagmus entirely eliminated. In the coal miners of Scotland, miners' nystagmus was common a few years ago, but it has become relatively unimportant in recent years owing to improvements in underground lighting.

Injuries from X-rays

Within a year of the discovery of x-rays in 1895 various ill-effects were reported. These included conjunctivitis, erythema, swelling and necrosis of the skin, alopecia, and chronic radiodermatitis. In 1897 attention was directed to the acute constitutional symptoms. In 1902 a case of cancer was recorded following chronic ulceration caused by x-rays. The first death recognized as due to the action of x-rays occurred in 1914. In 1922 it was estimated that 100 radiologists had died from malignant disease due to their occupation. Deaths occurred among radiologists exposed to x-rays before the importance of adequate protection was realized, and unfortunately still occur.

So far nearly all the victims have been research workers, radiologists, laboratory assistants, technicians, and nurses. Cases of industrial origin did not occur until later, because the use of x-rays in industry began later. Industrial workers, unlike professional workers, are often quite ignorant of the possible dangers of the apparatus used, so that industry is now, in respect to the use of radioactive substances, about where medical practice was in 1914. There is, however, no reason why the lamentable history of the pioneers in the medical field should be repeated. The human experiments have been made, the tragic results of carelessness demonstrated, and the measures necessary for adequate protection are known and available to anyone who cares to learn them.

X-rays may be used in industry for the detection of defects, cracks, and blow-holes in castings, of defects in alloys from faulty mixture, of erosion in cables or gas cylinders, and of defects in reinforced concrete or in weldings. X-rays are also used to sort fresh eggs from stale, to reveal mineral adulterants in vegetable

foods and weevils in grain. For the examination of metals and all thick objects very penetrating rays must be used.

Injuries which follow *a short single exposure*, or perhaps several exposures, may vary enormously in intensity. In mild cases there is simply a transient reddening, lasting a few days and followed by scaling and loss of hair. If the burn is deeper, blisters appear which may be serous or purulent, and the condition resembles that following a scald but is less acute and slower to heal. Sometimes the process, instead of disappearing in a few weeks, penetrates to the deeper layers of the skin and to the subcutaneous tissues, with the formation of a leathery slough, surrounded by a brawny indurated swelling with ill-defined limits. The process is exceedingly slow and obstinate, and has a tendency to progress and to resist treatment in a remarkable way. It is at times very painful.

In *chronic x-ray dermatitis* the changes in the hands begin round the base of the nails as a peculiar erythema and gradually increase. Transverse and longitudinal ridges appear on the nails, which become brittle, assume a characteristic dirty brown appearance, tend to separate from the matrix, and eventually thicken and form shapeless masses. The skin becomes uniformly red and atrophied; small warts appear, increase in size and number, and, when situated over the knuckles, crack and cause much pain. Later the dry thickened skin shows telangiectases, absence of hair, paronychia, and ulcers which are slow to heal and prone to break down. The hair follicles and the sebaceous and sweat glands completely disappear in cases of long standing. The freedom of the palms of the hands may be due to the naturally thicker skin there, but the greater liability to exposure of the backs of the hands and fingers is probably the more important factor. The lesions are, as a rule, slowly progressive. Post-irradiation telangiectases, which have been regarded as compensatory for obliteration of the vessels in the corium, usually appear within two years, and sometimes in the absence of an initial erythema; in some instances the interval between irradiation and the appearance of telangiectases is prolonged, even to 15 years. If exposure is continued the lesions may progress to involve the tendon sheaths and joints. There may be intense pain,

of which the severity is out of proportion to the size of the lesions; it is caused by the exposure of nerve-endings.

Squamous-celled carcinoma is almost always the form of malignant disease which has followed excessive x-ray exposure and long-continued x-ray dermatitis in man. Although most often seen in radiologists and manufacturers of x-ray apparatus (Plate 14), x-ray carcinoma may also occur in patients who have undergone treatment by x-irradiation. The interval between the onset of chronic x-ray dermatitis and the appearance of malignant disease varies from 3 to 27 years. The average of 35 cases was 7 years. The age incidence from 35 to 50 is comparatively early, that of ordinary carcinoma of the skin being between 55 and 58. The most frequent site of the growth, which is not uncommonly multiple, is on the backs of the hands and fingers, and the hand more exposed appears to be the one more severely affected, the left in radiologists and the right of those engaged in the manufacture of apparatus. Among radiologists carcinoma usually develops in an ulcer, less often in keratotic areas. The predominating symptom is pain, which may be constant and very severe, and has been ascribed to invasion of the terminations of nerves by the growth and to neuritis. Occasionally basal-celled carcinoma results from x-irradiation. In one case a basal-celled carcinoma of the scalp appeared 18 years after epilation for ringworm. A case has also been recorded of multiple basal-celled carcinomas on the trunk of a radiographer.

Constitutional symptoms only became prominent after the introduction of deep x-ray therapy, in which massive doses of deep penetrating rays were given. Severe constitutional symptoms may occur. They are nausea, uncontrollable vomiting, sometimes with haematemesis, diarrhoea, with the passage of blood, abdominal pain and distension, fever up to 104° F., restlessness, profound prostration, progressive cardiac failure, small rapid pulse, and dyspnoea. When death has occurred it has usually taken place about the fourth day from the onset. Both animal experiments and necropsies of human victims show that the application of x-rays to the abdomen may result in necrosis of the intestinal mucosa. As long ago as 1905 unsuspected sterility was found in 18 persons who had for various periods

been exposed to x-rays. The acute degenerative changes in the testes are followed by fibrous atrophy. Anaemia occurs in x-ray workers. After small doses of x-rays, the lymphocytes are first increased in number, then diminished. The red cells may also be increased at first, but anaemia sets in later and may become extreme. In patients who recover, the anaemia is slower to disappear than is the leucopenia. True aplastic anaemia does not occur. The possibility of exposure to radium should always be excluded before attributing what appears to be aplastic anaemia in an x-ray worker to x-irradiation.

In the *prevention* of these ill-effects extensive use is made of lead, lead glass, lead rubber, lead bakelite, barium bricks, and barium concrete. Although such measures have no strictly legal recognition, powers of inspection and approval have been placed in the hands of the National Physical Laboratory. A service has been organized whereby the radiation received by a worker can be assessed by means of the blackening of a small wrapped photographic film worn on the coat lapel during working hours for a week. If the film, when developed, indicates a higher dose than 0·3*r* per week the laboratory immediately follows up with an inspection of the department concerned. The results of film tests carried out on several thousand radiological workers from 1943 to 1948 indicated that about 80 per cent received less than 0·1*r* per week from external sources of radiation. Repeated periodical blood counts must be carried out in all doctors, nurses, research workers, students, technicians, and workshop employees exposed to x-rays.

It is important that the industrial physician should plan measures for the protection of employees against the dangers of x-irradiation, for in large manufacturing establishments there are x-ray departments for diagnosis as extensive and as much used as the average hospital department. Here the problem may include protection of workers in neighbouring rooms, for unless walls and floors are very thick or are rendered impermeable by the use of lead or barium concrete, x-rays may pass through them and cause injury.

The *treatment* of x-ray carcinoma is radical excision of a finger, or diathermic coagulation under local anaesthesia, or by

radium. Slight degrees of anaemia recover on removal from exposure. In more severe degrees iron must be used, and when this fails blood transfusion is necessary.

Injuries from Radioactive Substances

The gamma rays of radium appear to have a greater tendency than x-rays to cause aplastic anaemia; this impression is supported by the experimental evidence that the penetrative gamma rays of radium reach the bone marrow more readily than do x-rays. Three fatal cases of aplastic anaemia were reported in the London Radium Institute in 1920, one in a nurse and two in laboratory assistants. The *late effects of internally deposited radioactive materials* in man were studied from 1925 onwards. Paints consisting of crystalline phosphorescent zinc sulphide, rendered permanently luminous by the addition of a very small proportion of insoluble sulphate of radium, mesothorium, and radiothorium, came into use about 1908. Such paints are applied to the figures of clocks and watches, and certain important parts of the machinery of aeroplanes. At two factories, one in New Jersey and the other in Connecticut, 38 deaths occurred among factory girls, chemists, and physicists. The girls affected introduced the paint into their mouths through the habit of pointing the brush between their lips and swallowed it for periods of from one to four or more years. The insoluble radioactive materials became deposited in the body to such an extent that even during life radioactive emanations could be detected in the expired air. Pointing the brush with the lips was prohibited in 1924.

After death, bone was found to be the tissue in which the materials had mainly accumulated. *Aplastic anaemia* resulted from the continuous bombardment of the haematogenous marrow by alpha particles, and it was found that these changes were quite different from those due to external irradiation with beta and gamma rays only. Radioactivity in the bones and teeth was demonstrated by autoradiography. The bones when placed directly on photographic plates produced impressions in as short a period as 3 days. By 1952 there had been 14 deaths from *sarcoma of bone* which had appeared on an average 23 years after

cessation of exposure to the luminous paint. In 3 cases deaths occurred from carcinoma of the paranasal sinuses, one of them 34 years after removal from exposure. The total amount of radioactive material necessary to produce fatal results is extremely small. It is sufficient for 0·01 mg. to be distributed over the whole skeleton to produce a terrible death years after it has been ingested. Radium is thus the most deadly poison known; tetanus toxin previously held the record with a lethal dose of 0·22 mg.

Dermatitis due to radium, isolated by the Curies in 1898, was reported in October 1900. Insufficiently protected tubes of radium salts kept in the waistcoat pocket for 6 hours produced reddening of the skin and within 10 days or so ulceration. Dermatitis has been reported in a number of persons engaged in making radium preparations, and less often in medical men. The ill-effects of ingestion of radioactive substances include severe anaemia, sometimes aplastic, necrosis of the jaw, spontaneous fractures, sarcoma of bone, and carcinoma of the paranasal sinuses. The changes in the blood have been referred to as *anaemia radiotoxica*. The red cell count may drop below one million and the haemoglobin below 20 per cent. Leucopenia, granulocytopenia, and thrombocytopenia all occur. Purpura is followed by more serious bleeding, such as menorrhagia, haemoptysis, haematuria, and retinal haemorrhages. Necrosis of the jaw occurs. It is similar to that produced by phosphorus, and is attributed to infection supervening upon changes in the bone. In certain cases necrosis of the jaw does not occur, but after a number of years generalized changes in the bones develop with deformity and sometimes spontaneous fracture, a condition known as *radiation osteitis*. Bone sarcoma occurs in 25 per cent of the cases of occupational mesothorium and radium poisoning.

Though medical practice is now almost safe so far as x-irradiation is concerned, matters are very different in the case of radium. There is no doubt that many people are affected by handling radium, chiefly by the gamma rays. Their penetrative powers are so great that it is not practicable to secure complete protection. In the case of persons who carry radium about, the weight of lead they can bear to carry only partly protects them. Surgeons handling radium are also ill-protected. In the case of a man using

120 mg. of radium in the treatment of carcinoma of the cervix uteri, protection is very difficult, as each time he has to handle the substance closely and carefully. In such circumstances the best protection is to keep the radium at as great a distance away as possible until it must actually be handled. It is obvious that ingestion or inhalation of radioactive materials in industry is highly dangerous, and that all occupations involving the handling of such substances should be strictly controlled and supervised. In the watch industry, outside New Jersey and Connecticut, the practice of pointing the brush with the lips is unknown, and no ill effects have been observed in other countries. In the luminous-dial painting industry *safety measures* fall into two groups. First, there are precautions designed to reduce unavoidable exposure to a minimum and to improve the general conditions of work. These include protective gowns, rubber gloves and aprons, lead-glass screens in front of the operatives, and local exhaust ventilation. Secondly, examination of the operatives for over-exposure is necessary. In routine blood examinations, leucopenia, granulocytopenia, or thrombocytopenia call for a change of occupation in the worker concerned. The hands must be repeatedly inspected for dermatitis. Workers must wear beta- and gamma-ray sensitive photographic films, and the radon appearing in the expired air must be repeatedly measured.

Like lead, radium has been shown to be stored largely in the bones. A course of treatment with calcium versenate may prove successful in reducing the amount of retained radium as indicated by the amount of radon in the expired air. For aplastic anaemia repeated blood transfusions will be necessary.

Injuries from Fission Products

Fission of the uranium nucleus has led to two industries, the one concerned with the development of bombs which explode with the force of a mere million tons of trinitrotoluene, the other with the peaceful production of electric power. The stable and *radioactive isotopes* are essentially by-products of work on *atomic energy*, but they are assuming increasing importance in research, therapeutics, and industrial processes, and radiation hazards in

peace-time are therefore not confined to establishments concerned with atomic energy research and production. Ionization may be produced directly by charged particles such as electrons or protons, or indirectly by the passage of uncharged neutrons, which cause ionization by collision with some of the atoms of the tissues.

Danger may arise from direct radiation or from the inhalation or ingestion of radioactive materials. Neutrons and gamma rays are exceedingly penetrating, alpha particles can be stopped by a sheet of tin foil and beta particles by a tenth of an inch of aluminium. Alpha and beta particles having entered the body can cause an immense amount of damage although absorbed in a comparatively small thickness of tissue. Radioactive substances which are particularly dangerous are those which are selectively retained in the skeleton. Thus uranium, thorium, plutonium, and the isotopes of strontium and yttrium are bone seeking. Since some of these elements are excreted very slowly they will irradiate both bone and bone marrow continuously for many years.

The essential effect of irradiation is cellular injury leading to tissue necrosis. The erythron, lymphoid tissues, and immature germ cells are the most sensitive, and highly differentiated tissues such as bone, nerve, and muscle are less severely affected. Haemorrhage results from vascular injury, thrombocytopenia, and possibly from the presence of an anticoagulant substance in the blood. Secondary infection with ulceration of the gut and urinary tract and acute haemorrhagic necrotizing pneumonia are common. Sterility may be temporary or permanent and radiation cataract may appear years after exposure. Little is yet known as to the carcinogenic and mutagenic effects of ionizing radiations. Contamination of residential areas by radioactive dusts might cause cancer, and the bone-seeking isotopes would constitute a special long-term hazard. An increased incidence of hereditary defects in future generations is also a danger.

The most destructive effects of an *atomic explosion* are due to blast and to the release of thermal radiation. Casualties from blast, flash burns, debris, and fire comprised 85 per cent of the deaths at Nagasaki and Hiroshima. Ionizing radiation arises in several ways during the explosion and includes neutrons, gamma

rays, and beta rays. A small proportion of the energy of the explosion appears as fast neutron radiation, which has great penetrating power but rapidly loses its energy in traversing matter. It is of secondary importance to the gamma radiation which is also highly penetrating. Beta rays come to rest completely after passing through a few yards of air. When an atomic bomb explodes high in the air, fission products are dispersed and contamination does not occur unless they are carried down by rain. In ground or underwater explosions there is a risk of direct exposure, ingestion, and inhalation. The risk diminishes rapidly during the first 24 hours and its total duration depends on the prevailing weather conditions, the half-lives of fission products, and other complex factors.

In *atomic warfare* casualties can be divided, according to the degree of severity, into 4 groups, but the clinical picture in all the victims may be complicated by the effects of burns and other injuries.

Group I. Following exposure to intense radiation, nausea, vomiting, and shock occur within a few hours, and there is progressive weight loss, fever, and diarrhoea, leading to death from toxaemia within 2 weeks.

Group II. With less severe exposure, symptoms do not appear until about the third week, when loss of hair, aplastic anaemia, pneumonia, and severe gastro-enteritis result in death in the majority within 6 weeks.

Group III. In those who survive beyond the sixth week aplastic anaemia may become chronic, although many will still die from pneumonia, enteritis, and other forms of secondary infection. Complete recovery is possible within 6 months, but weakness and fatigue are common during convalescence.

Group IV. In mild cases leucopenia, diarrhoea, and loss of hair may be the only abnormalities.

Protective measures are necessary in atomic energy establishments, plutonium factories, and research laboratories. Sources of intense radiation such as reactors and cyclotrons are surrounded by massive concrete walls from 3 to 8 feet thick. Inhalation of radioactive gases is prevented by the use of gas masks or more elaborate air-conditioning devices, and protective clothing is

worn to prevent direct contact. When materials are exceedingly active they are encased in screened cells and are manipulated by remote control, using periscopes and mechanical hands (Plate 16) sometimes under the control of stereo-television. Pollution of the atmosphere with radioactive dusts is prevented by means of filters fitted to the stacks, and contaminated waste water is rendered inactive in special delaying and treatment tanks before it is released into the rivers and seas. Many forms of monitoring devices are used to ensure the effectiveness of precautions. All workers carry film badges, fountain-pen monitors, or other forms of electrometers; they also undergo medical examination with blood counts at regular intervals. The radiation levels of the environment are estimated with continuously running dust monitors, Geiger counters, and other instruments which are either portable or fixed. The inspection and supervision of workers is best accomplished by a special *health-physics team* which might employ 70 inspectors to deal with 1500 workers.

Occupational Bursitis

Chronic bursitis is one of the oldest occupational disabilities known. It occurs in those whose work involves pressure, friction, or repeated slight blows over a bursa. Increase of fluid then occurs in the bursal sac and a local swelling results. The olecranon bursa may be affected in students, bricklayers, and miners (miners' elbow), the ischial bursa in lightermen and weavers (weavers' bottom), the pre-patellar bursa in housemaids (housemaids' knee), potmen, clergymen, and nuns (nuns' bursitis), and the subacromial bursa in bricklayers (hod-carriers' shoulder). Connective tissue has the capacity to produce new bursae if they are required on account of oft-repeated but unusual types of movement in the body. Such adventitious bursae may be found over the vertex in market-garden porters (Covent Garden hummy), over the seventh cervical spine in fish porters (Billingsgate hump), over the upper part of the shoulder and clavicle in bricklayers, dustmen (dustman's shoulder), and timber porters (deal-runners' shoulder), and over the external malleolus in tailors (tailors' ankle).

Chronic bursitis usually produces a slowly enlarging, painless swelling, and the patient seldom pays much attention to it unless some complication occurs. Contusion of a bursa already distended with fluid may cause either a sudden increase of fluid or bleeding into the sac. In either case there is an acute increase in swelling, accompanied by pain. Where pyogenic infection of a bursa occurs, there is increased swelling accompanied by redness of the overlying skin and acute pain. In some cases of chronic bursitis, a portion of the sac wall may become calcified and then pain is likely to be persistent.

A patient with chronic bursitis seldom seeks treatment unless the bursa becomes painful or its size makes it inconvenient or unsightly. Fluid may be aspirated from the distended sac although it usually returns within a few days. When there is an acute infection, incision into the sac with drainage may be indicated. When chronic bursitis continues to be annoying, excision of the sac may be necessary. However, it must be remembered that with the removal of the bursa the normal protection to the underlying bony prominence has also been removed.

The Beat Disorders of Miners

The greatest sufferer from bursitis is the coal miner, who is liable to the so-called beat disorders. The man who works at the coal face is known as the coal-getter. He undercuts the seam of coal by a process known as bottom-holing, in which he lies on his side with his head thrown back and cuts the coal with a horizontal swing of the pick. Owing to the constrained attitude necessarily adopted, there is pressure on certain points, and the bursae over the patella and the olecranon may become enlarged and the surrounding fasciae thickened. Where mining is mechanized, continued kneeling may be necessary, especially in districts where the seam at the coal face is only 2 feet thick. Thus the highest incidence of beat knee is in cuttermen, prop-drawers, and conveyer-turners, who have to move about the coal face on their knees.

The bursae are liable to get infected, and for this reason the Industrial Injuries Act recognizes two types of beat disorder –

subcutaneous cellulitis and *acute bursitis*. The lesions are legally defined as subcutaneous cellulitis or acute bursitis over the elbow (beat elbow), and subcutaneous cellulitis or acute bursitis arising at or about the knee (beat knee). The subcutaneous tissues of the hands may become thickened owing to the pressure of the pick. Infection of these tissues gives rise to subcutaneous cellulitis of the hand (beat hand). Thus the aetiology of beat hand, which is always a cellulitis, differs from that of beat knee and beat elbow. Beat hand is not confined to miners; firemen stoking boilers, especially in ships, may suffer from subcutaneous cellulitis of the hand. In the beat disorders, cellulitis commonly arises from the extension of a staphylococcus infection of a hair follicle into the adjacent subcutaneous tissue. It is unusual to find a cut or abrasion which has afforded entry to the infecting organism.

The *symptoms and signs* are redness, swelling, heat, and pain. At first the skin is unbroken but pits easily. Generally, suppuration occurs with or without involvement of the adjoining bursae or tendon sheaths. In the case of the knee or elbow the course of the disease is usually benign and without sequelae, the patient being well in 6 weeks or even less. On the contrary, in the case of the hand the infection may involve the tendon sheaths and cause serious disability, sometimes with permanent maiming. It follows that an important aim in treatment must be to secure free movement of the wrist and fingers.

Since 1924 the *incidence* of the beat disorders in coal miners has increased more than tenfold. Although it was hoped that the increasing use in coal mines of machine cutters would greatly reduce the incidence of these conditions, the contrary has occurred, partly because the new methods of mining involve more kneeling. In the coal mines of Great Britain the use of knee-pads to reduce trauma, although widespread, has failed to stop the steady increase in the incidence of beat knee. It is probable that pads increase the liability to follicular infection because of maceration of the skin beneath them. Coal particles inevitably accumulate under the pads and reduce protection against trauma. The application of hardening agents to the skin is of doubtful benefit. Supervision of men at risk, and especially of those adjudged susceptible, in order to detect and treat infection while

it is still confined to the neighbourhood of the follicle, offers perhaps the most practical approach to the problem. An important therapeutic measure in most cases is temporary transfer to work where there is no kneeling to do.

Occupational Diseases of the Skin

In all industrial countries the incidence of occupational dermatitis is high. In Great Britain it accounts for most of the total annual injury benefit for all prescribed diseases or injuries which are not accidents. It is not difficult to appreciate the distress and anxiety among the unfortunate victims, as well as the length of disablement which is likely to result, or to recognize their lessened security on their return to work. Incapacity from this cause represents so much lost time and waste of health and money, for occupational dermatitis is largely preventible.

The causes of occupational diseases of the skin can be classified under four headings:

(i) Physical factors – pressure, abrasion, moisture, desiccation, heat, cold, light, x-rays, and other rays.

(ii) Plant products – leaves, stems, sap, roots, bulbs, flowers, fruits, vegetables, wood dusts, resins, and lacquers.

(iii) Living agents – bacteria, viruses, fungi, helminth parasites, insects, and mites.

(iv) Chemical substances – inorganic acids and salts, hydrocarbons, oils, tar, pitch, anthracene, and dyes.

In the case of chemical substances two different types of action on the skin are possible. These substances usually act as *primary irritants* when by virtue of their alkalinity or acidity, or from their degreasing, dehydrating, oxidizing, or reducing properties, they upset the skin on first exposure of all persons provided there is sufficient concentration and length of exposure. But some chemical substances, usually of complex organic molecular structure, or else simpler substances capable of combining with protein to form complex antigens, give rise to *sensitization dermatitis*. With such substances the first exposure is harmless, but if it is continued or repeated after a variable period of time, sensitization develops and subsequent re-exposure even to minute

quantities is followed by dermatitis. It is, of course, possible for a chemical substance to act both as a primary irritant and as a sensitizing substance in the same patient.

The chief causative agents responsible year by year for disability can be classed in the following order: alkalis, sugar, oil, chromium salts, turpentine, dyes, chemical substances, friction, petrol, dough, acids, paraffin, French polish, nickel compounds, and rubber accelerators. Similarly the occupations chiefly involved are as follows: dyers and calico printers, engineers, labourers, metal platers and polishers, bakers and confectioners, French polishers, painters, leather workers, chemical workers, rubber workers, sugar confectioners, textile workers, oil refiners, printers, sugar refiners, pottery workers, flour workers, and biscuit makers.

The elucidation of the cause in a given case often depends upon persuading the patient to describe what he does in his own words, which, if he is a technician, may be far from simple. A man's nominal work does not always indicate his risk. A workman calling himself a fitter may have been exposed for the whole of one day to *para*nitrochlorbenzene while repairing the plant used to make this substance in a chemical works. A maintenance engineer may one day handle water-pipes, another day a coal conveyor, and a third day a still caked with quinine residues. The diagnosis of his skin eruption is made by patch testing, but one only arrives at the need for this by particularity in questioning. Knowledge of irritants encountered in apparently innocuous occupations is of importance. Thus dermatitis in letterpress printers was traced to the presence of a minute proportion of formaldehyde added to an adhesive paste with the object of checking the growth of moulds. It is unsafe to forget that some men have two occupations and some have hobbies not free from cutaneous hazards.

The result of the application of external irritating agents to the skin is the appearance of a reaction varying in all degrees from mere discomfort to itching, smarting, or burning, and through stages of redness, punctate, or confluent up to a pustular, scaly, warty, or malignant ulcerative reaction. Most occupational dermatoses originate in the stomata, in hair follicles, or in the

folds of the skin and spread from these sites. In the great majority of the plant dermatoses pinhead vesicles predominate. Sometimes the causal irritant is identified because the dermatitis is associated with some other more characteristic lesion, such as *chrome holes*, *salt holes*, *soda holes*, *lime holes*, or *powder holes*.

Deductions may sometimes be made from the site of origin. For example, the dermatoses of lime, sugar, metal, and tulip juice have a predilection for, but are not always confined to, the nail areas. It is of some interest to note which hand is first and more affected, for if in a left-handed man the left hand is the first to show a skin lesion and remains throughout the worst part affected and the last to heal, it is difficult to dissociate the left-handed eruption from the left-handed man's occupation. It must be recognized that in workers in wet processes, notably dyers, chemical workers, and sugar refiners, the feet and legs are often first or alone affected. An irritating dust, as opposed to a liquid, will usually show its effect first on the face and neck, as do certain wood dusts, copra dust, and barley. The site of the lesion may be determined by local sweating, on the forehead especially, where caps are worn – namely, in woodworking, flour-packing, and dough-mixing. The thighs may be affected from irritation caused by oily overalls and trousers worn by fitters, engineers, and metal workers. Oil folliculitis is, of course, easy of recognition, but then it is not strictly a dermatitis.

Many cases of trade dermatitis are caused, not by the substances encountered on the job, but by their removal by degreasing agents and other substances harmful to the skin. The worst offenders amongst such cleansers are washing soda, soda ash, chloride of lime, paraffin, petrol, naphtha, turpentine, methylated spirit, and trichlorethylene. One such substance commonly used to remove dyes from the skin is a mixture of bleaching powder and soda ash known as *chemic*. Unless all trace of this is removed, preferably under running warm water, it may set up alkali dermatitis. The French polisher likes to use strong aqueous solutions of washing soda to remove the stains from his skin. The stronger the solution used, the quicker does he finish his toilet. Half a pound of washing soda to a gallon of water is of sufficient strength for his purpose. Painters use turpentine to

clean paint off their skin; cotton-seed oil should be used instead.

The risk is not confined to industry but extends to the home, where the domestic worker constantly uses soap and synthetic detergents, and may have her hands immersed in water for hours at a time; the housewife thus becomes the victim of *dishpan hands*. In addition, hot alkaline cleansing solutions are in frequent use, strong solutions of soda, quick chemical cleansers, and even chloride of lime are employed to save time, and strong or dilute acids are used to clean lavatory basins and closet pans. Metal polishes, furniture polishes, and leather preservatives are sold extensively to the housewife. Quick-drying enamels and paints, varnishes, special rapid paint renovators, disinfectants, insecticides, artificial plant manures, and storage batteries are in daily use in the home and may all cause dermatitis.

The sensitizers or allergenic chemical substances affect only a small percentage of people exposed to them; a few of them by sufficient exposure can produce sensitization in everyone. The tendency to hypersensitivity may be inherited. A complete list of sensitizing chemical substances would be a very long one; they include coal-tar products, explosives, photographic developers, dye intermediates, dyes, rubber accelerators, insecticides, oils, resins, synthetic resins, and plasticizers. Examples are phenol, picric acid, aniline, hydroquinone, crystal violet, hexamethylenetetramine, nicotine, tung oil, pine resin, urea formaldehyde, and dioctylphthalate. During 1948 nurses, doctors, and dispensers handling streptomycin began to be sensitized to this substance, and by 1952 the numbers affected in some areas caused anxiety. To a less extent penicillin has the same effect. It seems that something between 1 and 5 per cent of nurses using antibiotics become sensitive to them and that a severe degree of sensitization to one antibiotic is frequently associated with sensitization to others. A very early sign seems to be swelling of the eyelids, which, especially in the case of streptomycin, may precede all others. The clear predominance of the hands, arms, face, and eyes among the sites affected seems to indicate the importance of local contact.

The breakage of ampoules, spillage of solutions, contact with swabs and with the patient's skin can all be avoided by careful technique and by wearing gloves. Air should not be expelled from

a filled syringe except into the phial. Industrial experience shows that, in most people, removal from contact at the earliest sign, followed by simple treatment and careful technique thereafter, prevents the development of sensitization and enables a worker to continue her employment. In severe cases desensitization should be carried out, especially where a highly trained nurse would otherwise have to give up her special job.

The various harmful agents which injure the skin are not without their effect on the nails. Electroplaters using nickel salts, and men handling lime, as well as those working with formaldehyde, *formalonychia*, all can be affected by acute ungual eczema. Onychomycosis of brewers due to brewers' mould attacks workers who clean the fermentation vats; the nails present longitudinal fissures with facets and crusty excrescences at the root. Exposure to harmful amounts of radium or x-rays causes the nails to be striated, fissured, and brittle. Paronychia occurs in sugar confectioners, in girls who peel and squeeze oranges and lemons, *limonene dermatitis*, and in those who sort and pack tulip bulbs or gather the crop of tulips in the spring, *tulip fingers*.

The plants, vegetables, fruits, wood dusts, and resins which produce substances harmful to the skin make a very long list. The workers affected are horticulturists, florists, gardeners, nurserymen, market gardeners, field labourers, pharmacists, perfumers, confectioners, fruit pickers, hop pickers, workers in canning factories, and those handling certain insecticides and lacquers. The plants causing dermatitis belong to such Natural Orders as the Liliaceae, Primulaceae, Anacardiaceae, Orchidaceae, and Iridaceae. The vegetables causing trouble are parsnips, celery, asparagus, spinach, and haricot beans; the fruits are oranges, lemons, tangerines, and tomatoes. Sometimes the origin of a plant dermatitis is not immediately apparent. Thus women shelling peas in Covent Garden market may show severe dermatitis from mayweed, the stinking camomile (*Anthemis cotula*) plucked along with the pea-pods. In some cases the plants are more irritant at certain seasons of the year. Often the poisonous substance remains active even when the plant is dried. The sap, stems, bulbs, leaves, and flower-heads of daffodils, narcissi, jonquils, tulips, and hyacinths may act as primary irritants

causing painful lesions of the fingers with seasonal recurrence. The condition may spread as an itching dermatitis of the hands with desquamation and deep fissures – *lily rash*. In the bulb-growing districts of the Scilly Isles and Cornwall this condition may disable so many workers as to cause serious dislocation of the industry in the early spring. Sorters and packers of tulip and hyacinth bulbs also may be affected.

Of the primrose family, the familiar wild *Primula farinosa* is seen in country districts all over Europe. Its near relative, the cowslip (*Primula veris*), has been known to set up violent dermatitis in cowmen and milkers after the udders of the cow have passed over the plants in dewy grass. *Primula sinensis*, *Primula arendrii*, *Primula mollis*, and *Primula contusoides* contain irritants, but the cultivated hot-house *Primula obconcica* is the worst of the family and affects 50 per cent of people exposed to it. The poison is a primary irritant and is contained in all the glandular hairs which cover all parts of the plant above ground. The rash is composed of numerous closely placed, small, shiny, red, punctiform papules. They are accompanied by much smarting and itching. Occasionally these lesions are associated with large blisters. A gardener or nurseryman who has shown himself very sensitive to the plant hairs or pollen of *P. obconica* may be unable ever to work again with this plant or indeed with others of the same Natural Order.

Of the plants which blister the skin, the stinging nettle (*Urtica dioica*) contains formic acid, and the blister plant (*Ranunculus acris*), the windflower (*Anemone quinquefolia*), and the Easter flower (*Anemone pulsatilla*) contain anemonine, an acrid alkaloid related to cantharidine. The irritating substances in celery, producing *celery itch*, and in the peel of oranges and lemons, affecting marmalade makers, are hydrocarbons of the terpene series. Although *Chrysanthemum vulgare* of our English gardens but rarely gives rise to dermatitis in horticulturists, nurserymen, and gardeners, other varieties of the species are irritant. Pyrethrum is obtained from *Chrysanthemum pyrethrum* and from *Pyrethrum cinerariifolium*, plants which grow in various parts of Europe, Asia, Africa, Australia, and America. Considerable disability is caused in Europeans who grow these plants on a large

scale in Kenya. A dermatitis occurs which is severe and incapacitating, idiosyncrasy is a marked factor, and the condition is further aggravated because the plant irritant causes photosensitivity. Pyrethrum powder is widely used as an ingredient of agricultural and cattle sprays and powders used to kill flies, mosquitoes, ants, and cockroaches. It obstructs the trachea of insects and paralyses their neuromuscular system.

Carpenters, sawyers, lumberjacks, workers in lumber yards, wood polishers, cabinet makers, shipwrights, and furniture manufacturers often develop dermatitis from contact with wood dust and occasionally suffer from constitutional symptoms as well. The substances responsible are the sawdust, the sap, and the polishings or the oil of the wood. East Indian satinwood (*Chloroxylon swientenia*) produces in cabinet makers a papulovesicular eruption with brawny swelling of the skin. The face and upper respiratory tract may be involved and headache and anorexia occur. In 1908 Legge described the effects on workmen of handling ebony (*Diospyrus ebenum*), satin walnut, teak (*Tectona grandis*), and olive wood as well as rosewood (*Dalbergia latifolia*), Cuban sabicu wood, West Indian boxwood, and partridge wood.

In 1905 the use of South African boxwood (*Gonioma kamassi*) in shuttle making for the Lancashire cotton industry caused widespread disability. It was found that the machinery used in making the wooden shuttles produced a dust, the inhalation of which led to drowsiness in the workmen so that they nearly fell off their benches. A trade-union secretary dispatched pieces of the wood to the Department of Botany in Liverpool University asking Professor Harvey Gibson what it contained to cause such an effect. An alkaloid was discovered in the wood which induced a gradual slowing of the heart-beat. It undoubtedly accounted for the symptoms complained of by the workmen. The application of local exhaust ventilation to the benches of the workmen did not entirely suppress the dust, and other types of wood had to be substituted.

Other irritant woods are Oregon pine, balsa wood, and cocobolo, which is made into bowls, handles for cutlery, and walking-sticks. Musicians occasionally suffer from dermatitis of the lips

from contact with mouthpieces made from grenadilla wood. Cocus wood, or cokus ebony, causes an eczematous eruption both in carpenters who saw it into blocks and in musicians who use flutes made of it. When new woods such as African hardwoods come on to the market, it is proper not only to have samples named by the Forest Products Research Laboratory, Princes Risborough, Bucks., but also to patch-test the skin of the patient with sawdust and shavings.

Of the animal parasites involving the skin scabies, pediculosis, and the effects of mites must be considered. Outbreaks of scabies in the camps of soldiers, miners, and lumbermen are well known. Prairie camps also can be infested, hence the name *prairie itch.* In soldiers, interdigital burrows are rare and the hands are often quite free from lesions of any kind. Penile lesions are found in the majority of patients, but, rather than suspect syphilis, the doctor must remember that scabies is commonly contracted as a venereal disease. Animal sarcoptes often infest man: burrows are not observed in animal scabies. Victims of the sarcoptes of the horse are either soldiers in mounted units, grooms, coachmen, or veterinary students occupied in dissecting horses which have suffered from generalized sarcoptic mange. The sarcoptes of the camel has been transmitted to soldiers in camel corps in Eastern campaigns. Cows with sarcoptic mange can infest farmers and dairymen with the sarcoptes of cattle, hence the name *dairymen's itch.* Pediculosis corporis is of the highest importance in military campaigns because body lice convey typhus, trench fever, and relapsing fever. Happily, impregnation of soldiers' shirts with dicophanum (D.D.T.) is highly effective against this parasite. In tramps and vagrants the combined effects of lice, dirt, and constant scratching of the skin give rise to *vagabonds' disease* or phtheiriasis. The whole of the surface of the body is deeply pigmented, and the epidermis is thickened and covered with scabs and crusts from secondary infection.

Dermatitis caused by mites is responsible for the names *grain itch, barley itch, copra itch,* and *grocers' itch.* Grain itch occurs in men unloading grain from ships, especially barley and cotton seed. The eruption is caused by *Pediculoides ventricosus,* a parasite which feeds on the grain moth. It attaches itself to man by its

claws and apparently introduces an irritant to the skin through its sucking discs. The incubation period is from 12 to 16 hours. The eruption consists of urticarial wheals, papules, vesicles, and pustules on the chest, arms, neck, face, and back. Copra itch occurs in dock workers handling copra, the dried kernel of the coconut. The cargo is infested with the mite *Tyroglyphus longior* which transfers itself to the patient and, after an incubation period lasting from 24 to 48 hours, gives rise to innumerable intensely itching papules all over the body. This parasite makes no burrow into the skin but dies, leaving its chitinous cover as an irritant. The cheese mite belongs to the same species. It attacks dockers, railway loaders, and grocers, hence the name *grocers' itch*. Men handling raw vanilla suffer from an itching eruption thought at one time to be due to a chemical irritant, hence the name *vanillism*. The pruritus has been traced to a mite of the species *Tyroglyphus*. An intensely pruritic skin eruption traced to *Carpoglyphus passularum* in decomposing figs has been named *fig-mite dermatitis*. It affects dock labourers unloading cargoes of figs, and grocers who handle them.

Differential diagnosis is of great importance. Long continuance of an alleged occupational dermatitis should make one very suspicious of its cause. The time of onset of symptoms must be ascertained precisely; a skin lesion may be due to the sun during a holiday and not to work. Past treatment must be known, for the present condition may be a dermatitis medicamentosa. Again, the patient may present himself in a phase of urticaria of endogenous origins, or the attack may coincide with a seasonal exacerbation of a long-standing prurigo. If the lesion does not soon heal when the patient is removed from the suspected cause, then the real disease may be an anxiety neurosis with itching as a symptom. Traumatic dermatitis does not recur when the cause is removed and reasonable time has been allowed for it to heal. Cheiropompholyx may give rise to difficulty in differential diagnosis. Patients suffering from this condition give a clear history of pinhead irritating water blisters cropping out between the fingers. These lesions may occur in a frail skin, not only as a reaction to chemical substances but also to climatic and emotional stimuli. Certain types of work will increase the severity of an

attack of cheiropompholyx; water and chemical substances of all kinds will aggravate the irritation, and remorseless scratching will begin, but if it be due to the patient's work, cure on removal will be complete.

The *treatment* of occupational dermatitis is largely preventive. It is obviously unwise to employ anyone whose skin shows abnormality. These include young persons and those with frail and thin skins. All persons with hyperidrosis, seborrhoea, xerodermia, or a defective peripheral circulation should be rejected. Unfortunately, in certain occupations a moist greasy skin appears to be more easily affected, while a somewhat harsh dry skin remains unaffected, and *vice versa*. Since injury to the skin, whether caused by friction or accidents, is a factor in the production of dermatitis a periodic inspection of exposed parts of the skin is advisable so that any injury may be treated. Selection of employees by pre-employment examination, frequent medical and prompt first-aid treatment are measures of importance in reducing the incidence of occupational dermatitis. Employees and supervisors should be instructed in the nature of the hazards encountered during work, in cleanly methods of work and how to take care of the skin. Increasing attention to factory hygiene, and particularly to plant design, are of vital importance so as to reduce the chances of irritant materials coming in contact with the skin. Of special preventive measures, the most obvious is protection of the hands by means of gloves. In handling strong acids and alkalis they are essential, but in other operations such as biscuit making, baking, and sugar confectionery, and in certain mechanical operations, they cannot be used.

Provision of protective clothing, and carefully chosen barrier creams, are important *preventive measures*, but, although they are often necessary, they should not be relied upon by themselves unless it is impossible to reduce the hazard by improvement of plant design. Adequate washing facilities are essential in factories where irritant materials are used, and it is necessary to stress their strict supervision when once provided. The correct application of barrier substances and the institution of washing facilities make it possible to eliminate irritant cleansing agents which often themselves cause skin disease. Protection is not without its own

dangers. Rubber gloves may be pierced and give a false sense of security (Plate 15). It is easy to contaminate the inside of a glove and it then holds the harmful substance in close, warm, moist contact with the skin for hours at a stretch. It is necessary to realize that it takes more trouble and a longer time to teach the worker the proper use of gloves than to teach a medical student or student nurse an aseptic habit in donning gloves for use in surgery. A workman handling mustard gas may burn the skin of his penis for want of understanding what happens when he touches the outside of his glove as he removes it to go to the lavatory. Equally, when he comes back he may introduce the irritant material into the glove. Sometimes a lathe operator wears a protective apron with a hole in it and then gets oil folliculitis on the skin of the abdomen. In wearing rubber high boots, the temperature of the feet is kept unduly raised so that they may perspire excessively, causing the skin to become sodden and therefore more liable to disease.

The *early diagnosis* of occupational dermatitis is of the utmost importance, so that the employee may be removed from contact with the causative agent. Any other course may lead to intractable skin disease associated with sensitivity to many agents. In the treatment of the disease, early removal from contact with the irritant substance and protection of the inflamed skin are cardinal principles. The benefits of rest and hospital treatment for severe cases are striking. It is sometimes necessary to transfer the affected person to work not exposing him to risk. Thus a gardener or nurseryman who has shown himself very sensitive to particular plants may be unable ever to work again with these plants. Valuable work in the prevention of skin diseases in factories has been carried out by Dr Sibyl G. Horner, H.M. Senior Medical Inspector of Factories to the Ministry of Labour and National Service.

Occupational Cancer

It has long been known that workmen in certain occupations are unduly liable to develop cancer. Further study has shown that the disease is caused by prolonged exposure to some substance

used in the particular occupation, and that the type of tumour produced is specific for that occupation. Compared with cancer of unknown origin, occupational cancer is a relatively rare condition, but it is nevertheless interesting and important. The various types of occupational cancer have several characteristic features in common. They appear only after long exposure in the related occupation. This latent period varies somewhat, but is usually from ten to twenty-five years. Workmen who have been exposed for a sufficiently long period are liable to develop a tumour many years after leaving their work. Their removal from the occupation is therefore no safeguard against the disease. The average age incidence is earlier than that for cancer in general, and is dependent upon the age at which the men enter the occupation and on the latent period necessary for the tumour to develop. The disease is almost invariably preceded by well-defined precancerous lesions, which are characteristic for each particular occupation. The localization and to a less extent the histological nature of the tumours are remarkably constant in any one occupation. Unlike ordinary forms of cancer, the tumours are frequently multiple.

It is possible that the study of occupational cancer has led to greater advances in our knowledge of the causation and prevention of tumour formation than any other line of inquiry. It is true to say that the suffering of victims of occupational cancer initiated the onslaught on the problem of cancer by chemical technique. Historically, the study of occupational cancer was begun by Percivall Pott in 1775 when he drew attention to soot as a cause of scrotal cancer in chimney sweeps. This was followed by the work of Butlin who in 1892 showed that pitch, tar, and mineral oil similarly caused scrotal cancer. It is of course the coal tar in soot which causes cancer of the skin in chimney sweeps. In 1915 Yamagiwa and Ichikawa produced cancer experimentally by painting the ears of rabbits with coal tar. In 1922 Leitch produced experimental tumours in mice by applying Scottish shale-oil to the skin.

Investigation naturally shifted to the chemical identification of the substances responsible. The application of fluorescence spectroscopy to the study of carcinogenic substances proved to

be the indispensable technique which led to success. Sir Ernest Kennaway became the inspired leader of a team which studied *chemical carcinogenesis* and in 1924 discovered the first cancer-producing hydrocarbon 1:2:5:6-dibenzanthracene, and then tracked down and isolated 3:4-benzpyrene from pitch. Research was then set going on the synthesis and testing of the hydrocarbons which were chemical variations of dibenzanthracene. Benzene rings were added and subtracted, alkyl groups were substituted in different positions in the nucleus, hexagon rings were replaced by pentagon rings, C was replaced by N and by S, and as a result of all this activity the number of compounds which have been tested for carcinogenic activity must now be counted in thousands. In 1943 3:4-benzypyrene was isolated from Scottish shale oil.

The sites in the body commonly affected by occupational cancer are the skin, the lung, and the bladder. In most cases we have no exact knowledge of the carcinogenic substances responsible for the lesions. In addition to causing cancer of the skin, tar and pitch may also cause dermatitis and conjunctivitis.

The occupations concerned in causing *cancer of the skin* have been extensively investigated by Dr Sydney A. Henry, at one time H.M. Medical Inspector of Factories to the Ministry of Labour and National Service. Those affected include chimney sweep, tar, pitch, anthracene, and creosote worker, briquette maker, shale-oil worker, gunsmith, oiling coolie, cotton-mule spinner, and automatic-lathe operator. Long exposure to any of these agents may result in localized new growths, either papillomatous or keratotic. Such new growths, seen typically in the bituminous shale worker, and at first apparently entirely benign in nature, may, if untreated, adopt at any time the active and unregulated mode of growth which marks malignancy. In those engaged in extracting oil from shale such growths commonly appear on the arms, but in users of this and other oils they may appear elsewhere, on parts more freely or more continuously contaminated, or on parts which are more susceptible or subjected to added irritation. Thus the cotton-mule spinner may develop an oil cancer on any part of the skin surface, but the classical site in his case is on the scrotum. Similarly persons habitually exposed to

tar, or pitch, especially in the form of fume and dust, may suffer from warty skin growths which, if untreated, tend later to ulcerate and to show signs of malignancy. The retort-man exposed to fume containing tar in a gas-works or coke-oven plant, the man in a tar-distillery, and the man using tar for net-fixing, or for making brushes or brattice cloth, or in boat-building are all exposed to the same risk: so too are the various pitch-workers – pitch getters, pitch loaders, road-makers, labourers in patent-fuel works, and optical lens makers. Any of these may show after some years of exposure to risk warty growths which occur most often on the eyelids, cheeks, chin, behind the ears, on the neck, arms, scrotum, or thighs. Where such warts appear and show malignant changes it is probable that such changes are regional rather than focal, for pitch warts are not uncommonly multiple, and they may recur several times in the same area of skin. But not all tar warts become malignant; those appearing within the first few months of exposure to risk commonly disappear, especially if the exposure ceases, while those which appear many years later may or may not show malignant tendencies. All are true neoplasms, but not all of them persist, and yet where one has already disappeared spontaneously another may appear and show almost from the beginning active and unregulated cell proliferation. Tar or pitch may thus initiate in an area of skin a neoplastic change while a man is at work, but malignant warts or carcinomatous ulceration may not appear until long after he has ceased to be exposed to risk. This latent period may be of some years' duration. Few cases of malignant ulceration of occupational origin become clinically evident under 30 years of age: commonly they occur between the ages of 55 and 65. Industrial skin cancers do not readily give rise to secondary growths, but where, in any part of the skin, a neoplastic change has once been initiated and become apparent clinically, any subsequent form of irritation may again precipitate proliferative changes of a cancerous nature.

In the patent-fuel industry where anthracite dust and coal-tar pitch are handled, inflammation and ulceration of the corneal conjunctiva, slow to heal, may result in permanent scarring and interference with vision. Similarly, workers in a pitch-bed are

prone to suffer from acute dermatitis of exposed skin surfaces, and may at the same time suffer from corneal lesions.

Although the occurrence of cancer of the skin among Scottish shale-oil workers was first recorded in 1876, 30 years were to elapse before the discovery of the same disease in Lancashire cotton-mule spinners. In 1906 S. R. Wilson, when appointed house surgeon to the Royal Infirmary, Manchester, noticed that the patients admitted suffering from cancer of the scrotum were not chimney sweeps but mule spinners. So struck was he by the fact that in 1910 he wrote an essay on the subject. This was submitted for a scholarship but was not published until 1922. Cotton-mule spinning necessitates frequent bending over a horizontal bar, placed some three feet above the floor and running the length of the spinning machine. It is always moist with lubricating oil thrown off by the spindles, and frequent contact with this bar results in the clothing over the upper part of the thighs becoming soaked with oil. The cancer occurs on the scrotum in about 70 per cent of cases, and on the hand, arm, face, leg, and foot in the remainder. The oil apparently most potent for its carcinogenic property is Scottish shale oil. The incidence of cancer of the skin in mule spinners is high, and this appears to be the result of a gradual change over about 1870 from animal and vegetable to shale oil for purposes of lubrication. The number of mule spinners in England is about 14,900, and 1989 cases of cancer had been reported by 1933. The average age among mule spinners who contract the disease is 52 years. Less than 3 per cent manifest the disease in dangerous forms under 20 years' duration of employment, while the average duration before it occurs is 40 years.

And now, with the question of mule-spinners' cancer still unsolved, the problem of the carcinogenic action of lubricating oils has again raised its head, this time in the engineering trades. Mineral oils are used in a wide variety of engineering processes, for example as lubricants, as coolants in metal-cutting operations, and as quenching agents in the tempering of metals and alloys. The most continuous and widespread skin contamination of workmen is usually to be seen in *bar automatic* shops, where groups of from 20 to 150 workmen may all be exposed under similar conditions.

The automatic machine-tool is designed to conduct a series of operations without removal of the work, for example, upon a long metal bar turning out some such object as a bolt.

These machines frequently work at high speed requiring large amounts of coolant upon the tool edges, and, as they are at present designed, much oil is thrown into the air as a fine spray which eventually settles on machines and floor to form a film. Not only is the operator exposed to this spray, but also in the course of his work he often has to adjust the tool and the metal while the machine is in motion. In spite of the splash-guards which are at present in use, gross skin contamination is usual. Since the machine operator's clothing soon becomes soaked through with oil, the skin on the thighs and genital regions as well as on the arms becomes very oily. In general, the degree of skin contamination with oil in bar automatic machine shops is very much greater than that to be seen in mule-rooms in the cotton industry. Early in their work 80 per cent of these mechanics become afflicted with oil folliculitis; those who have been exposed for many years develop on their arms multiple hyperkeratoses and in 1950 one case of carcinoma of the scrotum was described. Better guarding of such lathes is urgently called for and should at once be demanded. The guards at present in use frequently have to be removed in the course of adjusting the cutting tool and many workers are careless about their replacement. It is possible that the fitting of automatic spring-back devices would ensure replacement of the guard.

Men working in the pitch beds of gasworks and in the patent fuel industry should be provided with goggles to protect the eyes from conjunctivitis and corneal ulceration. Protective clothing, a double locker system, baths, and washing facilities must be provided for workers in pitch, tar, anthracene, creosote, and shale oil, and for mule spinners and machine operators exposed to cutting oils. Barrier creams applied before work appear to be of little value. All these workers must be educated as to the danger of their occupation and should also be instructed how to minimize it. Regular periodic medical examination is long overdue. In compensation for death alone the cotton industry must antici-

pate paying £10,000 a year until it is realized that this sum could be considerably reduced and life spared by timely periodic examination. Voluntary examination is bound to lead to evasion, especially from the desire to conceal a condition situated, as it so often is, on the scrotum.

The doctor and the engineer should co-operate in devising apparatus which will prevent contamination of the skin with oil. This has been done for the spinning mule by fitting a device which holds a felt strip in contact with the mule spindle, thus preventing oil-splash. Cancer of the skin in industry will be got rid of in years to come either by the elimination from tar and mineral oils of carcinogenic substances or by their neutralization and destruction. It was shown in 1929 that the carcinogenic activity of certain oils is much reduced or completely removed by extraction with sulphuric acid, by oxidation, and by reduction. Later it was found that the method of solvent extraction had the same effect. Modern methods of oil refining are bringing within reach the possibility of the commercial production of non-carcinogenic oils. By agitation of oil distillates in liquid sulphur dioxide, aromatic fractions are extracted from petroleum distillates, leaving an oil which is mainly aliphatic in composition. This process has made practicable the production of non-carcinogenic *white oils* for use in the cotton industry. Much has yet to be learned before analogous developments can be applied economically to engineering oils.

The prognosis of carcinoma of the skin due to tar or mineral oil is extremely good provided that the patients attend early. In such cases treatment by radon or by flat plates of radium easily removes the whole neoplasm in one application. A large gas-works in London sends all its cases of pitch warts to the Radium Institute. A radon seed containing 1·5 millicuries of radon sheathed in platinum is inserted under the surface of the papule, the needle puncture being sealed with collodion. The local skin reaction is slight, so that patients can continue at work during and after treatment. After 5 days the seed is expressed. Such patients must be kept under observation for many years in order that further papules may be dealt with as they arise.

The industries concerned in causing or possibly causing cases

of *cancer of the lungs* are uranium mining, work at coal-gas retorts and coke ovens, nickel mining and the nickel industry, the chromates-producing industry, the asbestos industry, and the manufacture of sheep dip. Adequate protection of the workmen in these trades raises many difficulties. Often the carcinogenic substances have not yet been identified. For example in the essentially dusty work of nickel mining and refining it is not clear whether nickel compounds are responsible for cancer of the lung or whether metallic arsenides in the ores should be blamed. Naturally strenuous efforts are made to suppress dust and fume at every stage of the processes concerned, but such efforts do not always mean success. Occasionally a fresh discovery brings new hope to a manufacturing trade, as for example the successful use of DDT as a sheep dip in place of the arsenical dips which have been in use for generations.

Workers employed in the manufacture of synthetic dyes are liable to papillomata and *cancer of the bladder*. It is unfortunate that the name *aniline cancer* was given to this disease, for subsequent work has shown that aromatic amines other than aniline are responsible. It is certain that the carcinogenic substances are among the dye intermediates; the finished dyes are harmless. Benzidine and *beta*-naphthylamine have both been incriminated by animal experiments which were continued up to 10 years, in part because the clinical evidence that they produced cancer of the bladder was incontrovertible. Strong suspicion based on clinical observation falls also upon two processes in the dye industry, namely those for the manufacture of magenta and of auramine.

Since 1920 it has been observed that the incidence of bladder tumours diminished considerably after the institution of various *protective measures* in the factories, such as general cleanliness in the workrooms, adequate exhaust ventilation for the removal of vapours, measures designed to diminish the evolution of fume and dust, mechanical transport of chemical products in closed containers and improvements in the personal hygiene of the workmen. Nevertheless, in the dye industry in various parts of the world, bladder tumours still occur in large numbers.

Early in 1947 the dyestuffs group of the Association of British

Chemical Manufacturers appointed a committee to organize a large research project on industrial tumours of the bladder. The first task was an accurate field survey of the industrial data. At the outset no doubt existed that men employed in the chemical industry ran a greater risk of developing tumours of the bladder than those in the general population. What was needed was a statistical analysis of the facts, so that opinions formed by personal observation could be substantiated or refuted, and steps taken to eradicate the disease from the industry without delay. The deductions were based on well-devised and cleverly used statistical methods.

By February 1952, 455 cases of bladder tumour had been found among a population of 4622 men in the British chemical industry. The great majority (87 per cent) were known to have had contact with aniline, benzidine, *beta*-naphthylamine, or *alpha*-naphthylamine. The men at risk were about 30 times more likely than the general population to die of tumour of the bladder. The risk affects men engaged in manufacture, usage, or purification of the chemicals, in this descending order of severity. There is no clear evidence against aniline itself, but magenta and auramine are suspect. Occupational tumours occur on an average 15 years earlier than spontaneous ones. The age at onset depends on the age at entry into the industry – the induction-time is nearly constant at 15 to 20 years – but, though the length of exposure profoundly affects the risk, there is no evidence that severity of exposure influences the induction-time. Individual susceptibility, the cause of which is unknown, is a distinct feature, as it is in laboratory animals.

In *curative treatment* workers exposed should be told both of the risk they run and of the nature of the symptoms. Routine examination and centrifugation of the urine should be carried out for red blood cells and exfoliated bladder epithelium. Soon after the first cases were discovered in 1895, facilities were provided in the Höchst factories for cystoscopic examination of every suspicious case. Routine cystoscopy should become universal in the dye industry throughout the world. The strongest argument for the routine use of the cystoscope in aniline dyeworks is that men have remained well for a number of years after cysto-

diathermy of a papilloma. Early diagnosis of all cases would ensure removal of every papilloma before it did any harm.

There is every hope that the research worker ultimately will rid the dye industry of this horror. The researches necessary are complex and difficult, but work is going ahead in many countries and has already achieved important results. In England circumvention of the carcinogenic effects of *beta*-naphthylamine has been planned by Dr Maurice W. Goldblatt, head of the Industrial Hygiene Research Laboratories of I.C.I. What is the plan adopted? First of all it is known that *beta*-naphthylamine almost certainly loses its power to produce cancer if it is sulphonated. In the dyestuffs industry *beta*-naphthylamine is required mainly in azo-dyestuffs and then in the form of sulphonic acids or their salts. Therefore industry has been instructed to meet all the more important uses of this amine by isolating it only as a sulphonic acid and never as the free base. But then some of the uses cannot be met by sulphonates, and other ways will have to be found for them. A similar plan must be devised for benzidine, and at the same time research workers must continue their experimental research for other carcinogens, such as those which affect men employed in the manufacture of magenta and of auramine.

Meanwhile such excellent surveys as that carried out by the dyestuffs group of the Association of British Chemical Manufacturers should stimulate other industries to institute investigations into their own hazards. Those who know the subject well have entered a plea for more accurate case records in industry, earlier diagnosis of occupational cancer, more field surveys, and further experimental work.

GLOSSARY OF TECHNICAL TERMS

AGGLUTININ. A substance in the blood serum of immunized persons which causes bacteria to become grouped together in clumps.

AGRANULOCYTOSIS. An acute blood disease in which there is a serious deficiency of certain colourless corpuscles in the blood.

ALDEHYDE. A family of chemical substances related to the alcohols.

ALIPHATIC CARBON COMPOUNDS. A great family of organic compounds in which the carbon atoms are arranged in open chains. The name arose because animal fats belong to this group.

ALVEOLI. Small air-containing spaces in the lungs.

AMAUROSIS. Partial or total blindness.

ANOREXIA. An absence or a loss of appetite.

ANURIA. Total failure to produce urine.

APHASIA. Loss of the power of speech from a defect of central control in the brain.

APLASIA. Failure of a tissue to develop or to perform its function fully.

ARGYRIA. A permanent grey-blue discoloration of the skin arising from the deposition of metallic silver.

AROMATIC CARBON COMPOUNDS. A great family of organic compounds in which the carbon atoms are arranged in closed rings. Many of them have a fragrant smell.

ASEPTIC. Free from septic matter.

ASTEREOGNOSIS. Loss of the sense through touch of the shape and consistency of objects held in the hand.

ATAXIA. Unsteadiness of movement.

AXILLA. The armpit.

BRUCELLA. The name of a particular species of bacteria.

BULLA. A large blister.

BURSA. A small lubricated pouch placed between movable parts to act as a buffer.

BYSSINOSIS. An affection of the lungs due to cotton-dust.

CACHEXIA. A very under-nourished condition.

CARCINOGENIC. Producing cancer.

CARCINOMATOSIS. Widespread cancer throughout the body.

CELLULITIS. Inflammation of cellular tissue.

CHEIROPOMPHOLYX. A skin disease with blisters on the palms.

CHELATING AGENT. A chemical substance which readily combines with a metal compound.

CHOLINESTERASE. A substance present in all body tissues which splits up acetylcholine into choline and acetic acid.

COMATOSE. In an unnatural heavy sleep or stupor.

CORNEA. Delicate, transparent structure in front of the pupil of the eye.

CORYZA. Cold in the head.

CYANOSIS. Blue discoloration of the skin from deficient oxidation of the blood.

CYTOPLASM. That part of the cell substance which surrounds the nucleus.

DIPLOPIA. Double vision.

DYSARTHRIA. Impairment of articulation from weakness of the muscles of speech.

DYSPNOEA. Difficult or laboured breathing.

ECTOTHRIX. A type of fungus found on the surface of the hair shaft in certain ringworms.

ECZEMATOUS. Pertaining to eczema, a watery or weeping type of inflammation of the skin.

EMPHYSEMA. Over-distension of the lungs with air.

ENCEPHALOPATHY. Disease of the brain resulting from certain poisons.

ENZYME. A substance formed in the body which accelerates some chemical reaction on which life depends.

EPITHELIOMATOUS. Pertaining to epithelioma, a malignant tumour primarily derived from the skin or mucous surface.

ERETHISM. Abnormal increase of nervous irritability.

ERYTHEMA. A superficial blush or redness of the skin.

ETHMOID. Relating to the ethmoid bone, the perforated bone in the nose.

EUPHORIA. A disproportionate sense of well-being.

FAECES. Excretion of the bowels.

FERRUGINOUS. Containing iron.

FOLLICULITIS. Inflammation of a group of follicles in the skin.

GANISTER. A hard rock used to make bricks which resist high temperatures.

GINGIVAL. Pertaining to the gum or gums.

GRANULOCYTOPENIA. An acute deficiency of certain colourless corpuscles in the blood.

GRANULOMATOUS. Pertaining to a type of inflammatory tumour which does not produce pus, that is the matter discharged when an abscess bursts.

HAEMATEMESIS. The vomiting of blood.

HAEMATITE. An iron ore, blood-red in colour.

HAEMOGLOBINURIA. Urine which is a clear wine-red colour because it contains haemoglobin, the oxygen-carrying pigment of the red blood corpuscles.

HAEMOLYTIC. Causing the breaking up of the red blood corpuscles, releasing the haemoglobin.

HAEMORRHAGIC. Characterized by excessive bleeding.

HEMIANOPIA. Blindness of one half of the field of vision.

HYPERIDROSIS. Excessive sweating.

HYPERKERATOSIS. Overgrowth of the horny layer of the skin.

HYPERPNOEA. Panting, excessively deep breathing.

INFARCTION. The formation of a dead area of tissue because of the obstruction of a blood vessel usually by a clot.

IONIZATION. Separation of substances in solution into ions.

KERATOSIS. Overgrowth of the horny layer of the skin.

LACHRYMATION. An excessive secretion of tears.

LEUCOCYTOSIS. An increase in the number of colourless corpuscles in the blood.

LEUCOPENIA. A decrease in the number of colourless corpuscles in the blood.

MALAISE. A general feeling of discomfort and uneasiness.

MENORRHAGIA. Excessive menstrual flow.

METHAEMOGLOBIN. A substance similar in composition to haemoglobin, but having its oxygen more firmly united with it.

METHAEMOGLOBINAEMIA. The presence of methaemoglobin in the blood.

MICROGRAPHIA. An abnormal handwriting with very small letters.

NECROPSY. Examination of a dead body.

NECROSIS. Death of tissue cells.

NEOPLASM. A tumour either innocent or malignant.

NEPHRITIS. Diffuse inflammation of the kidneys.

NEUTROPENIA. Too few colourless corpuscles in the blood.

NYSTAGMUS. Oscillatory movement of the eyeballs.

OEDEMA. Accumulation of fluid in the cellular tissues leading to swelling (dropsy).

OLIGURIA. Scantiness of the urine.

OPISTHOTONUS. A spasmodic rigidity of the body in which the trunk is thrown backward and arched upward.

OVICIDE. Any substance poisonous to the eggs of insects, plants, or animals.

PAPILLOMATOUS. Of the nature of a papilloma, an epithelial tumour, a wart.

PAPULAR. Consisting of a papule, a small, solid elevation of the skin.

PARAESTHESIA. A morbid or altered sensation, a tingling in the skin.

PARAPLEGIA. A symmetrical paralysis of the legs.

PARENCHYMATOUS. Pertaining to the cellular functioning elements of an organ.

PARESIS. Slight or incomplete paralysis.

PARONYCHIA. Inflammation about the fingernail.

PETECHIAL. Characterized by small pinpoint, purple spots of haemorrhage under the skin.

PHAGOCYTE. A cell possessing the property of absorbing.

PHOTOPHTHALMIA. Disorder of the conjunctiva or even the retina of the eye caused by intense light.

PHTHISIS. A wasting or consumption. Pulmonary tuberculosis.

PNEUMOCONIOSIS. A disease of the lung from inhalation of dust.

POLYMORPHONUCLEAR. Having a nucleus deeply lobed or so divided that it appears to be multiple; a characteristic of some of the colourless corpuscles of the blood.

POLYNEURITIS. A disorder of many nerves at once.

POLYURIA. The passage of abnormally large amounts of urine.

PROGNOSIS. A forecast as to the probable result of an attack of disease.

PRURITUS. Intense itching.

PSITTACOSIS. A virus disease of parrots communicable to man, marked by high fever and pulmonary involvement.

PSYCHONEUROSIS. The state of mind in which a person has difficulty in adapting himself to his experience with the result that he suffers from physical symptoms or mental distress.

PURPURA. Minute haemorrhages into the skin appearing as purple spots.

PUS. Dead white cells of the blood; the matter discharged when an abscess bursts.

PYREXIA. High temperature, fever.

PYROLUSITE. Manganese dioxide.

RETROBULBAR. Pertaining to the back of the eyeball.

RHONCHI. Rattling noises in the bronchial tubes heard through the stethoscope.

RODENT ULCER. Malignant ulcer which gradually involves and eats away the soft tissues and bones. It generally occurs upon the face.

SARCOMA. A malignant tumour differing technically from cancer but producing similar effects.

SCOTOMA. A dark spot in the field of vision.

SCHIZOPHRENIC. Pertaining to or affected with schizophrenia, a split mind.

SEBACEOUS. Pertaining to the oily secretion of the skin.

SEBORRHOEA. A disturbance of the sebaceous glands marked by the occurrence of an excessive oily discharge.

SEPTICAEMIA. An infection characterized by the presence of bacteria in the blood.

SEPTUM, NASAL. The partition separating the two nasal cavities.

SEROLOGICAL. Pertaining to the clear part of the shed blood, the serum.

SERUM. Blood from which all the fibrinogen and corpuscles have been removed, rendering it incapable of clotting.

SIALORRHOEA. Salivation. Excessive flow of saliva.

SQUAMOUS. Scaly.

STAPHYLOCCOCUS. A species of bacteria.

STERNUTATORY. A gas or vapour which causes sneezing.

SYNOVIAL. Pertaining to synovia, the fluid which is contained in joint cavities, bursae, and tendon sheaths.

TACHYCARDIA. Fast action of the heart.

TELANGIECTASES. Red spots formed in the skin by dilated capillaries.

THIOARSENATE. A compound containing sulphur and arsenic.

THROMBOCYTOPENIA. Decrease in the number of blood platelets.

TRAUMATIC. Caused by an injury.

TRISMUS. A spasm of the muscles of mastication; lockjaw.

URTICARIA. An itching nettle-rash on the skin.

VESICANT. A blistering agent.

VESICLE. A small blister on the skin.

VOMER. A thin plate of bone forming the posterior portion of the septum of the nose.

WHITE CELLS. The colourless corpuscles of the blood.

XERODERMIA. An abnormal dryness of the skin.

YTTRIUM. A very rare metal.

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WORLD HEALTH

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Health, physical, mental, and social, should be everyone's birthright. How far this is from the truth and what deep considerations are involved in making it possible, is the concern of this book. The subject is vast and the issues that are raised by it are immense; but we must face them if mankind is to gain the strength and happiness which health can bring.

The main problems are now social rather than technical. Science has given us many powerful weapons with which to control the bacterial world as well as the means to provide ourselves with an environment in which health is possible; these must be put to the fullest use everywhere. But this cannot easily be done without an understanding of much deeper issues which the book discusses – geography, beliefs and customs, family life, population, occupation, town living, hospitals, food, and industrialization. Such social considerations include also the problems of administration – without which techniques and wisdom together will still be unable to bring health to the masses.

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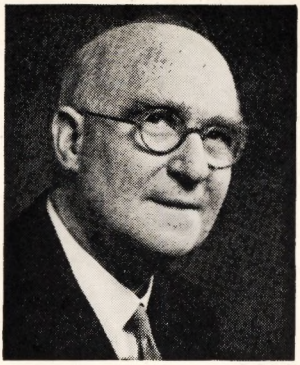

de Jongh

Donald Hunter, C.B.E., M.D., F.R.C.P., is senior physician to the London Hospital, Whitechapel. He was born in London in 1898, entered the medical school of the London Hospital in 1915, and qualified as a doctor in 1920 after service with the Royal Navy in the First World War. He was elected to the staff of his hospital as assistant physician in 1927. Dr Hunter served for many years on the Pharmacopoeia Commission and on the Poisons Board, and in 1944 he became founder-editor of the *British Journal of Industrial Medicine*.

His introduction to the problems of health in industry came in 1925 when he went to Harvard to work on lead poisoning with Dr Joseph C. Aub in the Massachusetts General Hospital, Boston. Back in Whitechapel he continued to make special studies of the dangerous trades. In 1943 Sir Edward Mellanby, Secretary to the Medical Research Council, asked him to accept the post of Director of a new Department for Research in Industrial Medicine at the London Hospital.

He is author of several books, including *The Diseases of Occupations*, 1957. Since 1945 he has visited many European countries and also Australia, Canada, the U.S.A., and parts of Africa, where he has had opportunities to see conditions of work in factories, mines, and shipyards.

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